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THE FACTORS LEADING TO DEATH IN OPERATIONS UPON THE GALL-BLADDER AND BILE-DUCTS *

BY GEORGE J. HEUER, M.D.

OF NEW YORK, N. Y.

FOR several months past I have been assembling and studying the cases treated in the surgical wards of the new New York Hospital since its opening, September 1, 1932, and with the view of determining the results of our surgical efforts. It seems desirable that I should do this; for if I can each year learn the reasons for our successes and particularly the causes of our failures it would seem that a more orderly progress toward the solution of the many problems which still confront us might well be made. I had intended to present a summary of our total experience in the past sixteen months but I find that to do so would take me beyond the time allowed me. By accident I first began a study of our experience with the diseases of the gall-bladder and biliary ducts (exclusive of malignant disease) and in connection with it assembled the literature with particular reference to the causes of death in operations upon the gall-bladder and biliary tract. It is based upon 200 cases treated in the New York Hospital between September 1, 1932, and March 1, 1934, 800 cases which I assembled from the records of the old New York Hospital between 1922 and 1932, and 36,623 cases assembled from American and European literature, a total of 37,623 cases. (Chart I.) In addition to these I have studied 1,066 cases specifically of acute cholecystitis (Chart II), and 5,815 cases particularly with reference to perforation of the gall-bladder.

Since this study was prompted by the fatalities which occurred in our own experience I shall begin its presentation by stating what this experience has been. Among the 200 cases operated upon for non-malignant diseases of the gall-bladder and bile-ducts, thirty-five were cases of acute cholecystitis subjected to cholecystectomy in the acute stage, with one death (mortality 2.8 per cent.); 134 were cases of subacute or chronic cholecystitis treated by cholecystectomy, with two deaths (mortality 1.4 per cent.); thirty-one were cases of chronic cholecystitis and common duct obstruction treated by cholecystectomy and choledochostomy with two deaths (mortality 6.4 per cent.). In the series of 200 cases comprising these three groups there were five deaths, a total mortality of 2.5 per cent.

An analysis of these five deaths, in four of which an autopsy was obtained, shows the following:

* Read before the New York Surgical Society, April 11, 1934.

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(1) A man, aged forty-one years, entered the hospital acutely ill with all the signs and symptoms of an acute cholecystitis. He was more toxic than usual and my impression was of an acute fulminating cholecystitis. He was operated upon about six hours after admission and at operation an acutely inflamed gall-bladder with one stone impacted in the cystic duct was found. A cholecystectomy was done. Following operation there was little change in his general condition; he remained seriously ill and toxic. On the third post-operative day he developed a bilateral acute parotitis; on the fifth post-operative day he showed an unquestioned bronchopneumonia; on the sixth post-operative day he developed generalized convulsions, lapsed into unconsciousness and died with symptoms of meningitis. An autopsy was not obtained.

CHART I

Gall-Bladder Operations from the Literature, Deaths and Mortality (Per Cent.)

Author and Reference	Number Cases	Number Deaths	Mortality (Per Cent.)
Blalock, A.: Bull. Johns Hopkins Hospital, 35:391, 1924	735	70	9.3
Smith, M.: Trans. Am. Surg. Assn., 51:287, 1933.....	1,053	71	6.7
Cattell, R.: ANNALS OF SURGERY, 89:930, 1929.....	890	41	4.2
Verbrycke: South. Med. Jour., 22:452, 1929.....	302	21	6.9
Johnson and Pearre: South. Med. Jour., 19:889, 1926..	488	47	9.6
Cave, H.: ANNALS OF SURGERY, 84:371, 1926.....	629	35	6.8
Hitzrot, J.: ANNALS OF SURGERY, 84:829, 1926.....	482	50	10.4
Judd, Parker: ANNALS OF SURGERY, 84:419, 1926.....	1,036	27	2.6
Bernhard: Bruns. Beitr. Chir., 150:83, 1930.....	4,557	209	4.3
Hotz: Arch. klin. Chir., 126:295, 1923.....	12,144	1,128	9.8
Siegmund: Deutsch. z. Chir., 230:359, 1924.....	850	46	5.4
Heuer, G.: West Va. Med. Jour., 26:1, 1930.....	135	11	8.1
Danzis: S. Clin. N. A., 6:1397, 1926.....	215	13	6.0
Davis: ANNALS OF SURGERY, 87:735, 1928.....	160	6	3.7
Sanders: ANNALS OF SURGERY, 92:375, 1930.....	500	20	4.0
Doran, <i>et al.</i> , ANNALS OF SURGERY, 98:330, 1933.....	200	14	7.0
Fowler: Am. Jour. Surg., 22:53, 1933.....	1,206	71	5.9
Stanton: Am. Jour. Surg., 8:1026, 1930.....	10,000	500	5.0
Santee: ANNALS OF SURGERY, 93:1156, 1931.....	333	21	6.3
Richter: ANNALS OF SURGERY, 88:187, 1928.....	418	23	5.5
Darner: Surg. Gynec., and Obstet., 37:579, 1923.....	290	29	10.0
TOTAL.....	36,623	2,453	6.6

(2) A man, aged sixty years, suffering from proven generalized arteriosclerosis with hypertension and myocarditis entered the hospital with symptoms of gall-stones. A diagnosis of chronic cholecystitis and cholelithiasis was made. At operation, which was technically simple, a cholecystectomy was done and the removed gall-bladder contained seventy stones. Signs of pneumonia appeared on the second post-operative day with symptoms of myocardial failure. There was a partial suppression of kidney function manifested by a high urea nitrogen. Death occurred on the sixth post-operative day. At autopsy there was a localized biliary peritonitis, a dilatation of the right ventricle, brown pigmentation of the myocardium, generalized arteriosclerosis and hyperæmia of the lungs, liver and kidneys.

(3) A man, aged forty-four years, entered the hospital with a markedly enlarged liver and deep jaundice. The pre-operative diagnosis was difficult and he was kept under observation some time in an attempt to differentiate between stone in the common duct and malignancy in the head of the pancreas. His icteric index was 121 on admission, subsequently rose to 140, but before operation was 80. Fluids and glucose were administered and he received 10 cc. of 5 per cent. calcium chloride daily. After eleven

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days of observation and study an exploratory laparotomy was undertaken. At operation a cholecystectomy was done and the exploration of the common duct showed multiple stones, sixteen of which were removed. The common duct was drained through the stump of the cystic duct. On the second post-operative day the patient vomited repeatedly, some of the vomitus containing blood, and had an elevated temperature; on the sixth post-operative day he vomited 10 cc. or more of bright red blood following which he rapidly passed into a condition of profound shock suggesting a massive hæmorrhage into the stomach. In spite of blood transfusions he failed to rally and died. Autopsy showed one stone in the common bile-duct near the duodenal orifice, which had not been removed at operation, obstructive biliary cirrhosis of the liver, chronic obstructive interstitial pancreatitis, hæmorrhagic pancreatitis with fat necrosis, multiple erosions of the gastric and œsophageal mucosa and hæmorrhage into the stomach.

(4) A man, aged sixty-two years, was admitted deeply jaundiced with a clinical history of common-duct obstruction. He presented in addition arteriosclerotic heart disease, hypertension and a mild grade of diabetes (arteriosclerotic). At operation a cholecystectomy was done and exploration of the common duct revealed two stones which were removed. The common duct was drained through the stump of the cystic duct. His post-operative course was marked by a low-grade temperature which fluctuated daily for fifty-three days, and by a persistently increasing diabetes. He was repeatedly examined, and the possibility of a subphrenic abscess was entertained but the diagnosis of this condition was never positively made. He died suddenly on the fifty-third post-operative day. At autopsy two very small stones were found in the common duct which had not been removed by operation, there was a chronic subphrenic abscess, a bronchopneumonia involving the right lower and left upper lobes, fibrosis of the pancreas, generalized arteriosclerosis, and cardiac enlargement.

(5) A man, aged eighty years, deeply jaundiced, was admitted with the clinical history and findings of common-duct obstruction due to stone or malignant disease. The surgical opinion, after study, favored a non-malignant obstruction. For his age the patient was considered in fair condition and was subjected to operation. At operation the gall-bladder was found to contain stones and the pancreas was enlarged and hard. A cholecystostomy was performed and twenty-five stones were removed from the gall-bladder. The common duct was examined but not opened. The gall-bladder was drained. The post-operative course was prolonged and unsatisfactory; nothing definitely was found to warrant further surgery. He died sixty-four days after operation. At autopsy there was found pneumonia (Friedlander type) with multiple pulmonary abscesses, a loculated abscess in the right pleural cavity, bronchopneumonia of the left lower lobe, pulmonary tuberculosis, and chronic pancreatitis.

In a consideration of the factors leading to death in these five cases we have to consider the operative treatment of cholecystitis in its acute stage, the consequences of operations upon the biliary tract in general, including infection of the peritoneum, hæmorrhage and shock, the post-operative pulmonary complications, and the cardiorenal complications. These and other factors concerned in the mortality following surgery upon the the gall-bladder and biliary ducts I shall attempt to classify.

A summary of 36,623 cases of gall-bladder and biliary-duct disease subjected to surgery shows a general mortality of 6.6 per cent. The mortality of individual surgeons or clinics, however, shows great variations, being in the hands of some as low as 2.6 per cent., in the hands of others as high as 10.4 per cent. (Chart I.) A summary of 1,066 cases of acute cholecystitis subjected to surgery shows a general mortality of 8 per cent., an individual mortality varying between 4.7 per cent. and 22.5 per cent. (Chart II.) A

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CHART II

Acute Cholecystitis; Operations in the Literature with Deaths and Mortality (Per Cent.)

Author and Reference	Number Cases	Number Deaths	Mortality (Per Cent.)
Miller, R.: ANNALS OF SURGERY, 92:644, 1930.....	200	27	13.5
Zinninger, M.: ANNALS OF SURGERY, 96:406, 1931.....	89	7	7.8
Mentzer, S.: Surg., Gynec., and Obstet., 55:709, 1932..	71	16	22.5
Judd and Phillips: Trans. Am. Surg. Assn., 51:292, 1933	508	24	4.7
Graham, H.: ANNALS OF SURGERY, 93:1152, 1931.....	198	12	6.0
Pratt, G.: Am. Jour. Surg., 22:46, 1933.....	45	10	22.2
Heuer, G.: West Va. Med. Jour., 6:1, 1930.....	56	3	5.0
Smith, M.: Trans. Am. Surg. Assn., 51:287, 1933.....	107	10	9.3
TOTAL.....	1,274	111	8.7

summary of 502 cases of gangrene with perforation of the gall-bladder subjected to surgery shows a general mortality of 46 per cent. and an individual mortality varying from 15 per cent. to 65 per cent. (Chart III.)

CHART III

Gall-Bladder Perforations from the Literature, Deaths and Mortality (Per Cent.)

Author and Reference	Number Cases	Number Deaths	Mortality (Per Cent.)
Blalock, A.: Bull. Johns Hopkins Hospital, 35:391, 1924	21	?	
Smith, M.: Trans. Am. Surg. Assn., 51:287, 1933.....	24	?	
Johnson: South. Med. Jour., 19:889, 1926.....	9	9	
Judd, Parker: ANNALS OF SURGERY, 84:419, 1926.....	2	?	
Siegmund: Deutsch. A. f. Chir., 230:359, 1924.....	8	5	62.5
Heuer, G.: West Va. Med. Jour., 26:1, 1930.....	18	?	
Danzis: Surg. Clin. N. A., 6:1397, 1926.....	1	1	100.0
Stanton: Am. Jour. Surg., 8:1026, 1930.....	32	32	?
Santee: ANNALS OF SURGERY, 93:1156, 1931.....	5	3	60.0
Alexander: ANNALS OF SURGERY, 86:765, 1927.....	20	?	
Mitchell: ANNALS OF SURGERY, 88:200, 1928.....	16	?	
McWilliams: ANNALS OF SURGERY, 55:235, 1912.....	6	2	33.3
McWilliams: ANNALS OF SURGERY, 55:235, cites.....	108	52	48.0
Gosset, Deplas: Jour. de Chir., 25:259, 1925.....	111	?	52.2
Darner, Cullen: Surg., Gynec., and Obstet., 37:579, 1923	3	2	66.6
Zinninger: ANNALS OF SURGERY, 96:406, 1931.....	16	?	
Miller: ANNALS OF SURGERY, 92:644, 1930.....	8	?	
Graham: ANNALS OF SURGERY, 93:1152, 1931.....	7	?	
Judd, Phillips: Trans. Am. Surg. Assn., 51:292, 1933..	68	?	
Mentzer: Surg., Gynec., and Obstet., 55:709, 1932.....	19	?	
TOTAL.....	502		

I have attempted to analyze the cause of death in this collection of cases. I find that the task has been extremely difficult due to the lack of accurate information, yet it is possible to arrange the causes of death under various headings and this I shall try to do.

(1) *Acute Cholecystitis, Gangrene and Perforation of the Gall-Bladder, Extracholecystic Abdominal Abscess.*—In a survey of the literature it becomes apparent that the acute conditions of the gall-bladder have contributed

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in no inconsiderable way to the mortality of gall-bladder and biliary-duct surgery. It is impossible from the present literature to get any accurate idea of the total number of cases of acute cholecystitis and its complications and it is impossible to estimate the total number of deaths which clearly have been due to them. It commonly has been stated by those favoring a waiting policy in acute cholecystitis that the occurrence of serious complications as gangrene, perforation and extracholecystic abscess is rare and that the mortality in gall-bladder and bile-duct surgery is conserved on the whole by waiting for the acute cholecystitis to subside. To determine the true frequency of the complications of acute cholecystitis and their dangers to the individual it is necessary to analyze, not the total cases of gall-bladder and duct disease, but solely the cases of acute cholecystitis. Unfortunately, the available information on this subject is unsatisfactory but certain figures are available.

In the New York Hospital series of 800 cases there were 106 cases which certainly were instances of acute cholecystitis and in this number are sixteen perforations and seven probable perforations of the gall-bladder, a percentage of 21. Judd and Phillips,¹ in 508 cases of acute cholecystitis, found sixty-eight with gangrene or perforation of the gall-bladder, a percentage of 13.4. Heuer,² in seventy-four cases of acute cholecystitis (Cincinnati series), found eighteen cases of gangrene, perforation or extracholecystic abscess, a percentage of 24; Zininger,³ in seventy-eight cases of acute cholecystitis, found sixteen cases of gangrene or perforation, a percentage of 20.5; Morris Smith,⁴ in 107 cases of acute cholecystitis, found twenty-four with gangrene, perforation and extracholecystic abscess, a percentage of 22.4. In the papers of seventeen authors I find the records of 502 perforations of the gall-bladder but I cannot accurately estimate the percentage this number represents of the cases of acute cholecystitis observed. It seems probable, however, that at least 20 per cent. of the cases of acute cholecystitis will have the complications of gangrene, perforation and extracholecystic abscess or peritonitis if a policy of inactivity toward the disease is adopted. The mortality in operations after gangrene and perforation have taken place is high. In the New York Hospital series of sixteen perforations and seven probable perforations there were eight deaths, a mortality of 34.7 per cent. This constitutes 11 per cent. of the total mortality in the series. In the experience of fourteen authors who have studied it, the mortality varies between 15 per cent. and 65 per cent., with an average mortality of 46 per cent.; and the mortality constitutes about 10 per cent. of the total mortality in operations upon the gall-bladder and biliary ducts (Chart 2). In certain articles, as for example that of Johnson and Pearre,⁵ the mortality from perforations alone constitutes 20 per cent. of the total mortality in a general series of cases. This is very largely a preventable mortality and has been due to an attitude of mind toward the treatment of acute cholecystitis on the part of the physician and surgeon. Fortunately, in my opinion, this attitude is rapidly changing. The recent publications⁶ show that an increasing number of surgeons are favoring operation in the acute stage of acute cholecystitis with which I have long been in

accord. The supposed dangers of operating in the acute stage have been over-emphasized. The removal of the acute non-gangrenous, non-perforated gall-bladder is usually not difficult and is attended by a mortality which in the hands of various surgeons has varied from 2 per cent. to 6 per cent. This mortality I am sure may be lowered and by an earlier attack upon the disease. The argument that the mortality in operations for chronic cholecystitis is less than that in acute cholecystitis is still true if one eliminates the mortality from gangrene and perforation of the gall-bladder. But from my point of view this mortality is chargeable to subacute or chronic cholecystitis rather than to acute cholecystitis; for it has resulted from the attempt to convert acute cholecystitis into the chronic form of the disease.

To sum up, the experience of surgeons who have contributed to the literature on the subject indicates that the complications of acute cholecystitis, including gangrene, perforation, extracholecystic abscess and peritonitis form a factor of importance in the deaths following operation upon the gall-bladder and bile-ducts. The mortality is largely preventable and in great part can be eliminated by operation in acute cholecystitis before these complications have occurred.

(2) *Errors in Technic and Complications Traceable to the Operative Procedure.*—A review of the material collected for study shows that errors in surgical technic, or if not these, then complications traceable to the operative procedure have been a factor of considerable importance in the mortality following operations upon the gall-bladder and bile-ducts. Again, it is quite impossible to state accurately how many deaths have been directly caused by these factors. In our own series of five deaths, I find that one death was due to acute bilateral parotitis and bronchopneumonia possibly the result of a cholecystectomy in acute cholecystitis. In the autopsy following a second death there was found a local biliary peritonitis, in a third a chronic subphrenic abscess and in the autopsies of two, common-duct stones had not been removed. In the last two the failure to remove all the stones from the common duct probably had nothing to do with the fatal outcome but in the first two the operative procedure contributed toward the fatalities. In the 800 cases of the New York Hospital between 1922 and 1932, peritonitis following operation and given as the major cause of death is recorded in eighteen cases, hæmorrhage in five cases and shock in four cases. These deaths form 37 per cent. of the total number of deaths. Scattered mishaps, such as evisceration, failure to remove stones, *etc.*, are occasionally recorded but are rare. In the 2,392 deaths in 35,373 cases gathered from the literature I find peritonitis given as the cause of death in 450 cases, shock in 173 cases, and hæmorrhage in 141 cases. To these should be added thirty-one deaths from post-operative ileus. These factors would, if correct as given, account for 33 per cent. of the deaths. But as I have stated in discussing acute cholecystitis and perforation of the gall-bladder, I have found it difficult to obtain accurate information. It is not clear, for example, in how many instances peritonitis was due to a gangrenous gall-bladder, to the spread of infection from an extracholecys-

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tic abscess, to the escape of infected bile from an open duct, and so forth. Nor is it clear whether in the instances of hæmorrhage, this hæmorrhage came from the liver, from a badly ligated cystic artery or was of the nature associated with jaundice. The figures show, however, whether strictly accurate or not, that the operative procedure, and the errors in technic and judgment committed during it, are factors still of importance in the mortality following operations on the gall-bladder and bile-ducts. To eliminate them is of course our natural aim. Aside from the slow meticulous performance of the operative procedure with the least injury to organs and tissues, the careful control of hæmorrhage, the prevention of soiling of the peritoneum by adequate protection and the use of suction, I have nothing to offer in a technical way except the drainage of the common duct through the stump of the cystic duct as first described by Halsted.⁷ This in our hands has been, I feel sure, a life-saving procedure in those cases in which the common duct has been opened and merits wider adoption.

(3) *Pulmonary Complications.*—Certainly to me one of the most disturbing features of surgery of the gall-bladder and bile-ducts is the high incidence of pulmonary complications. In the five deaths of our series, an extensive bronchopneumonia confirmed by physical signs and X-ray demonstration was present in the case of acute cholecystitis operated upon in the acute stage of the disease. In this case an autopsy was not obtained. In the four additional patients who died, autopsy showed pneumonia as the major cause of death in two. Of five deaths, therefore, pulmonary complications appear to have been the chief factor in causing death in three. These are the findings in the deaths but do not include the larger number of pulmonary complications from which patients have recovered but which nevertheless have been a source of great potential danger. These include seven instances of pneumonia, five of massive atelectasis, and three of pulmonary embolism and infarction. In an analysis of the deaths in the old New York Hospital cases I find pneumonia listed as the cause of death in twelve cases, and pulmonary embolism in seven cases. In four additional cases pneumonia was found at autopsy in association with a varying grade of peritonitis. In this series, therefore, 25 per cent. of all deaths were due to pulmonary complications. Of the 2,392 deaths in 35,373 cases in the literature, pneumonia is given as the cause in 317, and pulmonary embolism in 137. Again, if these figures are correct, pulmonary complications have contributed 20 per cent. to the total mortality in surgery of the gall-bladder and bile-ducts. It would appear, then, that post-operative pulmonary complications form one of the important factors in the mortality following surgery of the gall-bladder and bile-ducts. They form a problem which in our experience is not yet solved. The careful administration of inhalation anæsthesia, whether ether, nitrous-oxide-oxygen or ethylene followed by over-ventilation of the lungs, fails to prevent them; nor has the use of local or spinal anæsthesia in our experience aided materially in preventing them.

(4) *Cardiorenal Disturbances.*—In the series of 200 cases which I have

studied arteriosclerosis of varying grade was almost a constant finding in patients over fifty years of age. Sixty-eight patients had hypertension indicated by a diastolic blood-pressure over 90, and fourteen gave a history of cardiac disease, of which ten had definite symptoms of decompensation. Of the five deaths in this series, one was due to myocardial failure with suppression of kidney function. In the 800 cases in the old New York Hospital series, 10 per cent. of the deaths appear to have been caused or largely to have been caused by failure of the heart. Of the 2,392 deaths in the 35,373 cases in the literature, 285, or 12 per cent., are ascribed to cardiorenal disturbances. It would appear that not only the constitution of the individual prone to gall-stone disease but also the chronic cholecystitis itself predisposes to arteriosclerosis, hypertension and myocarditis. Early diagnosis and treatment of the affections of the gall-bladder and biliary ducts would certainly avert some of the deaths following operations for these affections. Careful study and pre-operative supervision by the surgeon of patients with arteriosclerosis, hypertension and cardiac disease may and probably do avert an occasional death after they have fallen into his hands. The problem, however, would seem to be a larger one and concerns the physician as well as the surgeon. In the 200 cases I have studied the average duration of definite symptoms of gall-stone disease was five and a half years, a period sufficiently long perhaps to provoke or to increase the changes in the vascular system so often seen in these patients.

(5) *Liver Insufficiency; Liver Death.*—The train of symptoms leading to death included under the terms of liver insufficiency and "liver death" is, I presume, familiar to you. Heyd,⁹ who so far as I am aware first called attention to it as a clinical entity, describes three types: In Type I the patient with simple cholecystitis without jaundice fails to recover consciousness after cholecystectomy or lapses into a semicomatose condition and with a steadily rising temperature and pulse rate dies in a period of thirty-six hours; in Type II, the patient with obstructive jaundice seems, after choledochostomy with drainage, to be progressing favorably for a few days, then presents evidence of cerebral excitation, becomes delirious, but later stuporous and then lapses into coma. Coincident with this change the escaping bile becomes less in amount and more watery in color and consistency; in Type III, less common than Types I and II, the patient with disease of the common duct and pancreas but without jaundice appears to be progressing satisfactorily for twenty-four to thirty-six hours after choledochostomy with drainage, then has a marked acceleration of the pulse, a fall in blood-pressure, a cessation of urinary secretion and exhibits cold, moist extremities. The clinical condition presented is similar to that of shock but occurs later than is usual in shock. Heyd⁹ suggests as the cause an overwhelming intoxication, probably of pancreatic origin. Transfusion and repeated infusions with ten per cent. dextrose in saline may save the patient.

Since Heyd's publications on this subject quite a number of papers have appeared both in this country and abroad. Schutz, Helwig and Kuhn¹⁰.

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describe a series of cases which on the fifth or sixth post-operative day showed a rise in temperature and pulse rate accompanied by abdominal distention and a progressive oliguria with the appearance of albumin, casts and often red cells in the urine. The patients became delirious and then comatose, the nitrogenous elements of the blood increased and the urinary nitrogen diminished. Nausea and vomiting were often severe and bleeding from the mucous membranes frequently became a prominent feature of the clinical picture. The condition then progressed to one of uræmia with almost total anuria, the patients dying with the clinical picture of uræmia. Autopsy showed quite consistently striking degenerative changes in the liver and kidneys. The liver showed leucocytic infiltration, necrosis and interstitial hæmorrhages or marked parenchymatous and fatty changes most marked about the gall-bladder fossa. The kidneys showed parenchymatous swelling and loss of normal markings and microscopically degeneration and necrosis of the tubular epithelium. These authors conclude that damage to the liver of traumatic or infectious origin results in the formation of a selective toxin which acts directly upon the kidneys producing degenerative changes in these organs. They consider their cases analogous to Heyd's "Type I." By other authors the pancreas is thought to be seriously implicated in the condition, by still others the liver alone through absence of glycogen.

It is not within the scope of this paper to attempt a full discussion of this condition called "liver death" nor could I discuss it intelligently having seen so little of it. I have never happened to observe a death in cholecystitis without jaundice as described by Heyd under his "Type I" in which the autopsy did not reveal conditions sufficient to explain the death otherwise; but I have recently observed a death closely resembling Heyd's "Type I" in a patient upon whom I performed a splenectomy for supposed Banti's disease but which subsequently proved to be a diffuse process in spleen and liver called by one pathologist a reticulosarcoma, by another Hodgkin's disease. She died in twenty-four hours with a steadily rising temperature and pulse rate but mentally clear almost to the end, and at autopsy failed to present anything in her chest or abdomen to account for her death. The clinical picture described under Heyd's "Type II" I have long been familiar with and have chiefly been impressed with the thin, watery character of the bile. As an interne more than twenty-five years ago, I recall that W. S. Halsted, my chief, commented repeatedly upon the danger to life of the patient who at operation presented a dilated common duct containing thin, watery bile and whose bile drainage subsequently became thinner and more watery. Since that time I have at times observed this condition and it has always been associated with a very high mortality. Presumably the sudden release of back pressure upon the liver by opening and draining the common duct causes in some way the suppression of liver function and this idea has led to attempts to gradually decompress the liver by certain technics in the drainage of the common duct. With Heyd's "Type III" I have had very little personal experience but that post-operative manifestations included under Heyd's "Type III" have long

been observed but perhaps not sufficiently emphasized seems certain. Again Halsted, in 1899,¹¹ describes a case of secondary operation for common-duct obstruction which some days after operation developed a very rapid and feeble pulse, vomited persistently, the vomitus containing blood, presented cold extremities and seemed certainly beyond the hope of recovery. Rather suddenly she improved and repeated infusions of salt solution were thought to have been instrumental in her recovery.

In an analysis of the 800 cases in the new New York Hospital series the suggestion of "liver death" is noted in eleven instances. These cases presented symptoms post-operatively which resemble those described in "liver death" but are without autopsy confirmation. Of the 2,392 deaths in 35,373 cases collected from the literature, so-called "liver deaths" are recorded in ninety-five or approximately in four per cent. of the deaths. At the present time the cause or causes of the condition are vague and not clearly understood; nor do we know as yet accurate tests to determine the extent of liver damage due to gall-bladder and biliary-duct disease. Graham's¹² experience with the use of iso-iodikon (dose 2.5 Gm.) would appear an encouraging step in the direction of a satisfactory test and he himself is disinclined to operate upon patients with a high dye retention. As a preventive measure the administration of dextrose pre- and post-operatively seems, in the light of our present knowledge, the most successful; and the use of calcium post-operatively as suggested by Graham¹² may be of value. The occurrence of the condition is another argument for earlier treatment of the affections of the gall-bladder and biliary ducts.

(6) *Miscellaneous and Undetermined Factors.*—In a study of our own series acute hæmorrhagic pancreatitis was present at autopsy in one case and in the 800 cases of the New York Hospital it was the cause of death in one case. In the literature I have assembled, pancreatitis is given as the cause of death in forty-eight cases, which represents 2 per cent. of the total mortality. In reading the histories covered by our studies a great variety of miscellaneous conditions have been assigned as the cause of death but because of their infrequency are of relative unimportance as factors leading to death in operations upon the gall-bladder and biliary ducts. In about 15 per cent. of all cases studied the factors leading to death could not be determined.

Summary.—In a study such as this the greatest good accrues to the individual who makes it; and I apologize if I have bored you with information with which you are already familiar. Quite independently of each other I have attempted to determine the factors leading to death following operations upon the gall-bladder and biliary ducts in two groups of cases, a group of 1,000 cases assembled from the records of the New York Hospital (Chart IV) and a group of over 35,000 cases collected from American and foreign literature. Aware throughout the study that the figures I obtained for various causes of death are only approximate, yet I find on comparing these figures in the two groups of cases that they are very similar. Thus the percentage of the total mortality from the complications of acute cholecystitis as gangrene

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and perforation in the New York Hospital series is eleven per cent., in the collected cases 10 per cent., that due to the consequences of the surgical procedure as peritonitis, hæmorrhage and shock in the New York Hospital series is 37 per cent., in the collected cases 33 per cent., that due to pulmonary complications in the New York Hospital series is 25 per cent., in the collected cases 20 per cent., that due to cardiorenal complications in the New York Hospital series is 10 per cent., in the collected cases 12 per cent. These are the major factors causing death and together account for approximately 80 per cent. of the deaths. Lesser causes of death are "liver deaths" (4 per cent.) and deaths due to pancreatitis (2 per cent.). In approximately 15 per cent. of the deaths, the causes of death form a miscellaneous group of conditions or have not been determined.

CHART IV

Causes of Death—New York Hospital, 1,000 Cases; Literature, 36,623 Cases

Gangrene and Per- foration		Conse- quences of Surgical Procedure				Pulmonary Complica- tions		Cardiorenal Complica- tions		Liver Death		Pancreatitis		Miscel- laneous and Un- determined	
		Peritonitis, Shock													
N.Y.H.	LIT.	N.Y.H.	LIT.	N.Y.H.	LIT.	N.Y.H.	LIT.	N.Y.H.	LIT.	N.Y.H.	LIT.	N.Y.H.	LIT.	N.Y.H.	LIT.
11%	10%	37%	33%	25%	20%	10%	12%	?	4%	?	2%	17%	19%		

These figures, inaccurate though they may be, so closely correspond in two groups of cases studied independently that I think they are of significance and suggest the direction our efforts should take in attempting to reduce the mortality following operations upon the gall-bladder and biliary ducts. An intelligent selection of cases for early operation in the acute stage of acute cholecystitis will, I am sure, largely eliminate the 10 per cent. mortality now occurring in the general run of patients with gall-bladder disease and the higher mortality in acute cholecystitis. So, too, more meticulous surgery combined with earlier diagnosis and treatment will reduce and, I think, greatly, the combined mortality from the consequences of the operation, including peritonitis, hæmorrhage and shock, from cardiorenal complications and from liver insufficiency, which in its totality accounts for approximately 50 per cent. of the deaths; and may reduce, chiefly by operating from one-half to one or more decades earlier in life the mortality from pulmonary complications. A consideration of the causes of death and their elimination cannot fail to make evident the importance of early treatment in diseases of the gall-bladder and biliary ducts. The difficulties which beset the surgeon during the operation and after largely are the results of deviations from the normal, the result of prolonged disease; the cardiorenal complications largely are the result of prolonged disease as is damage to the liver resulting in the so-called liver deaths. The deeply jaundiced patient with common-duct obstruction with his low vitality, his non-resistance to infection, his liver damage, and his tendency to hæmorrhage is the result of prolonged disease; and he will

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die all too frequently notwithstanding the best surgery. Our efforts toward lowering the mortality in gall-bladder and biliary-duct disease should be in two directions, to persuade the physician and patient that safety lies in early surgical treatment and to perfect our own judgment and technic in the performance of this surgical treatment.

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IMPORTANT FACTORS IN THE SURGICAL TREATMENT OF CHOLECYSTITIS *

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THERE has been no improvement in the mortality from gall-stone disease during the past decade, as shown by the data of the Metropolitan Life Insurance Company, which correspond closely with the general results published for the United States registration area. During this period, there have been marked advances made in our knowledge and methods of treatment of these cases. Amazing mortality results have been obtained in some of our large centres. Henry Cave¹ reported, eight years ago, the splendid mortality of 6.8 per cent. in 515 cases of biliary disease at the Roosevelt Hospital. Since that time, The Mayo Clinic has reported a 3 per cent. mortality in 703 operations for benign lesions of the gall-bladder and bile passages and 2 per cent. mortality for cholecystostomy and cholecystectomy in 656 cases (1932 report.) Evarts Graham, after carefully eliminating the bad risks by his liver-function dye test (phenol-tetra-iodo-phthalein), showed a mortality of .4 per cent. in 224 cases operated upon in 1929-30-31. Yet one of our large and good metropolitan hospitals in its published surgical reports for 1928-1932, inclusive, shows 460 operations with fifty-two deaths, or 11.3 per cent. mortality. Another in the 1932 report shows over 13 per cent. mortality in fifty-two cases. This is much too high.

We have prepared this paper in the hope that it may stimulate further analyses of gall-bladder mortality in our hospitals and prove a useful summary of our present knowledge of the care of these cases. Pulmonary and cardiac complications are the ones most to be feared.

Overholt² has demonstrated the following facts:

- (1) Chest expansion is diminished 75 per cent. after operations in the upper abdomen.
 - (2) The excursion of the diaphragm is diminished 50 per cent. after abdominal operations.
 - (3) Vital capacity is diminished 64 per cent. after operations in the upper abdomen and 15 per cent. by a tight binder.
 - (4) Morphine, by its relief of pain, increases vital capacity.
- The value of carbon dioxide in compelling deep breathing is generally accepted.

Hugh Cabot³ says: "My own experience in the past year seems to indicate that the routine administration of carbon dioxide has very importantly diminished the number of post-operative lung complications. I incline to the view that the post-operative use of carbon dioxide is the most important single contribution to the ultimate safety of anaesthesia which has been made in many years."

* Read before the New York Surgical Society, January 24, 1934.

Eliason⁴ points out the danger of undressing patients, particularly men who have been used to heavy clothing, and putting them in bed in a thin cotton nightgown.

Gordon Heyd⁵ has made a valuable contribution by emphasizing that glucose is good for both acidosis and alkalosis and the prevention of sudden death following operations upon the gall-bladder and ducts.

Experimentally, normal function of the liver seems to occur with only 15 per cent. of normal hepatic tissue if the animal is kept on a diet high in carbohydrates—mostly milk and syrup—but, if meat is given, cirrhosis with ascites will develop and the animal will die. This is a striking indication for pre-operative and post-operative treatment, when one considers that almost every case of cholecystitis shows evidence of an accompanying hepatitis.

When first seen, these patients with cholecystitis are immediately classified as acute or chronic. We believe that the acute cases should be operated upon as promptly as is consistent with safety. This usually means within a few hours. Walton⁶ says: "Since it is impossible to determine whether the inflammatory changes will progress or resolve, an operation should always be advocated in the early stages. If carried out within the first twenty-four hours, it will be as free from danger as the corresponding one performed for acute appendicitis."

Three years ago, one of us (H. F. G.⁷), analyzed 198 consecutive operations for cholecystitis. Twenty patients were operated upon within forty-eight hours of the onset of acute symptoms. There were no deaths from early operation when acute cholecystitis was the only disease present at the time of operation. The one patient who died had an acute pancreatitis already present before operation.

These early operations were usually simple ones. The post-operative complications were few. The days in the hospital and the dressings were few. There were no ruptured wounds and the cost to the patient was low.

"Watchful waiting" was formerly considered the proper policy in acute cholecystitis, but a survey of the recent literature shows a steadily increasing tendency among our best surgeons to operate early. Pratt⁸, reporting the cases of acute suppurative and gangrenous cholecystitis from Wayne Babcock's service for a period of thirteen months, mentions twenty-three cholecystectomies performed within twenty-four hours after admission to the surgical service. All recovered. About 25 per cent. were approximately sixty years of age. In the entire series of forty-five cases, there were no deaths when the operation was performed within forty-eight hours after the onset of the colic. Stone and Owings⁹ have practiced cholecystectomy on all forms of acute gall-bladder disease over a period of years, without the loss of any of them. Judd and Phillips, Lund, Miller, Homans, Royster and Finney at the last meeting of the American Surgical Association all expressed themselves definitely in favor of early operation. We think that the time has now arrived when one must justify delay in removing an acutely inflamed gall-bladder.

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The cases of chronic cholecystitis require more study. In many of them, a more or less extensive rehabilitation must be attempted. We will assume that a correct diagnosis has been made.

Allen Whipple¹⁰ has called attention to the greatly increased post-operative morbidity when upper respiratory infections were present before operation. One such patient in our series was sent home for two weeks to return later for a successful cholecystectomy. One patient had twenty-one teeth removed and another eight teeth removed before operation.

A woman, aged fifty-seven, five feet five inches tall, weighing 177 pounds, short of breath and with a systolic murmur over the cardiac area, was referred back to her family physician for cardiac treatment and reduction in weight. Five weeks later, after a loss of seven pounds in weight, an acute attack rendered operation imperative. A successful cholecystectomy was done for acute suppurative cholecystitis with stones.

A man, aged sixty-eight, was seen at his home in consultation, suffering from acute cholecystitis. His pulse was almost imperceptible and his heart sounds were poor. Non-operative treatment was advised. After three days, he entered the hospital with a palpable mass in the gall-bladder region, but we delayed another four days, because of his alarming cardiac condition. Digitalis by mouth and glucose solution intravenously were followed by progressive improvement in his general condition, so that he finally had a cholecystectomy, without post-operative complication.

Transfusions may be necessary for anæmia and chronic jaundice.

Calcium chloride or lactate and glucose solutions are indicated when post-operative hæmorrhage is feared.

Every jaundiced patient should have an estimation of the coagulation and bleeding time.

We have had no personal experience with Evarts Graham's functional liver test, but in his hands, it seems to have been 100 per cent. perfect, for he went one entire year without an operative gall-bladder death. Our only question is whether this test is too perfect and denies operation to some who ought to have it.

All patients who are to have a gall-bladder operation should have a high carbohydrate diet, without meat, immediately preceding the operation, unless vomiting or diabetes prevents. This means cereal, fruit juices, hard candy, tea with sugar, oatmeal gruel with sugar, crackers, toast, baked potato, etc.

Every case should receive at least one infusion at 500 cubic centimetres of 10 per cent. glucose solution intravenously before operation. It is usually given immediately preceding the operation. Patients suffering with acute cholecystitis who are good operative risks may need no further preparation than the glucose intravenously and cleansing of the skin area.

Operative technic seems to be well standardized. We wish merely to emphasize the advantages of cholecystectomy without drainage whenever possible. One anatomical variation is important to bear in mind. An accessory right hepatic duct is sometimes found emerging from the liver in the fossa of the gall-bladder and running a variable course to enter the common

bile-duct. If unrecognized and not ligated, it is a source of great danger, as death from biliary leakage may ensue.

I quote from E. Starr Judd¹¹: "Closure of the wound without drainage is a perfectly safe procedure if the surgeon is certain that the cystic duct and artery have been securely tied, if the fossa of the gall-bladder has been carefully sutured, and if the operative field is dry." "Closure without drainage favors healing and lessens the incidence of postoperative hernia." De Courcy¹² shows a mortality of 2.7 per cent. in a series of 36 cholecystectomies without drainage. Goldish and Gillespie¹³ have shown that undrained cases average 3.6 days shorter time in the hospital than the drained ones.

We have analyzed a series of 155 consecutive cholecystectomies, without drainage, that have been done by a few of us who have been associated in this work for some years. There have been nine deaths, giving a mortality of 5.8 per cent. We believe that freedom from the pain caused by a drain is an important factor in permitting deep breathing, following an operation.

It is obvious that gall-bladder operations tend to diminish the depth of inspiration, with a resultant tendency to pulmonary congestion, atelectasis and interference with the circulation, especially of the right heart, due to diminished negative pressure on the great veins of the thorax. Large doses of the barbiturates for hypnosis during the post-operative period will also cause shallow breathing.

To prevent these ill effects, the following are important: (1) Eliminate the binder entirely. (2) Avoid large doses of the barbiturates. (3) Use morphine in moderate doses for relief of pain. (4) Use the sitting position to aid the accessory muscles of respiration and take the weight of a heavy, fat abdomen off the diaphragm during inspiration. (5) Use inhalations of carbogen (10 per cent. or 15 per cent.) for five minutes every two hours for at least twenty-four hours, even if it hurts. (6) Teach each patient to breathe deeply every fifteen or twenty minutes "To avoid pneumonia" and nearly every one will do it voluntarily. (7) Use a shoulder cape or blanket to prevent chilling, especially at the zero hour when the steam is off. If you will investigate, you will find that this is not an infrequent occurrence. In summer, excessive perspiration, open windows and drafts increase the danger of surface chilling.

The glucose solutions should be continued intravenously and subcutaneously after operation until fluids are taken freely by mouth. In patients, who had not previously responded well to glucose alone, some remarkably good recoveries have been obtained at The Mayo Clinic, by adding ten to twenty Gm. of sodium lactate to the glucose solution administered intravenously. If a cough with thick, tenacious sputum develops after operation and bronchial occlusion with atelectasis is feared, steam inhalations and liquefying expectorants should immediately be used.

Our attention and interest were first centred upon this problem in September, 1932. In the sixteen months since that time, we have done sixty gall-bladder operations on the First Surgical Service at the Methodist Hospital with four deaths, a mortality of $6\frac{2}{3}$ per cent. In our first forty-

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eight operations, we had two deaths, a mortality of about 4 per cent., while there have been two additional deaths in our last twelve cases.

These results may not be striking, but we are convinced that a definite, well-planned routine is essential for the successful operative treatment of patients suffering from cholecystitis.

A detailed account of the deaths is added to invite criticism.

CASE I.—September 20, 1933, L. A., a woman, aged sixty-seven, was sent in to the Medical Service at the Methodist Hospital by the surgeon who later operated upon her. Seven years before, she had suffered from an apoplectic stroke and dizziness had persisted since that time. For three weeks she had been ill with nausea, vomiting and dizziness. Her blood-pressure was 230/130 on admission.

She remained on the medical service nine days, during which time she was nauseated and vomited daily. Her blood chemistry was normal. No relief was obtained from medical treatment, so a cholecystectomy was requested and performed.

An adherent gall-bladder, containing a stone near the cystic duct, was removed. On the following day, the patient seemed stronger and on the second day after operation the pulse was regular and slow, the color was good and the abdomen was soft. As evening approached, however, the pulse became very irregular, there was a general convulsion and the patient suddenly expired. (October 1, 1933.)

Known to be a desperate risk, the question here was one of mortality statistics versus an effort to relieve an otherwise incurable case.

CASE II.—January 9, 1933, a woman, aged fifty-eight, was admitted to the Methodist Hospital. She had been ill for twenty-four hours with severe epigastric pain. On admission, there was a distended abdomen, blue lips, distant heart sounds and an almost imperceptible pulse. The diagnosis was acute cholecystitis or pancreatitis. She was considered an impossible operative risk. Eleven days later, operation revealed a perforated gall-bladder full of stones, with an abscess outside and a swollen pancreas.

The abscess was packed with iodoform gauze and a tube inserted.

The post-operative course was quiet for about three weeks, except that great weakness and listlessness were noted. The patient seemed tired all the time. Then a low-grade, irregular temperature appeared, and a secondary drainage operation was performed, re-opening the old wound, which looked unhealthy. Later vomiting commenced and gradual failure. Death ensued on February 25, 1933—thirty-six days after operation. An autopsy showed a general peritonitis with pus in the pelvis and flanks. The pylorus was adherent to the liver and the lesser peritoneal sac was filled with 50 cubic centimetres of yellow pus. Several small abscesses were present in the fat behind the pancreas and right kidney.

CASE III.—February 23, 1933, M. R., a woman, aged thirty-five, was admitted to the Methodist Hospital. For five weeks she had suffered from repeated attacks of pain in the right upper quadrant, jaundice, chills and light colored stools. Operation was performed six days later. A thick-walled, white gall-bladder containing one stone was removed and the common duct, which was dilated, was drained by a T-tube.

There was a considerable quantity of sanguinopurulent discharge from the wound. On the eleventh day, after operation, there was a profuse hæmorrhage from the wound. Transfusions were given as follows: 1,000 cubic centimetres on the eleventh day, 900 cubic centimetres on the sixteenth day, and 900 cubic centimetres on the nineteenth day. On the twenty-eighth day, operation revealed a pelvic abscess and intestinal obstruction. A colostomy was done. Cultures from the wound showed *B. coli*, non-hæmolytic streptococci, Gram-negative extracellular diplococci. There was gradual failure and death April 22, 1933—fifty-three days after operation.

Autopsy showed a subacute general peritonitis and pelvic and mesenteric abscesses.

This patient, unlike the previous one, was not lethargic but was a high-strung, excitable Spaniard.

CASE IV.—This is the only "doubtful" case from the standpoint of lack of drainage. November 27, 1933, A. E. B., a female, aged sixty-one years, entered the Methodist Hospital. She had been ill for nearly a month with recurring epigastric pain and vomiting. For forty-eight hours before admission, the pain and vomiting had been more severe. On admission, her temperature was 101.8°, pulse 96 and respirations 24. The leucocytes were 14,750 and the polymorphonuclears were 82 per cent.

Five hundred cubic centimetres of a 10 per cent. solution of glucose were given intravenously and three hours later a forty-five-minute cholecystectomy without drainage was performed. A large, thick-walled gall-bladder was removed intact. When opened, after the operation, it was found to contain muco-pus. The area surrounding the gall-bladder showed no evidence of infection, but the liver showed marked hepatitis. Her temperature became normal on the fifth day after operation and remained so until the fourteenth day. Convalescence was smooth, but the patient seemed despondent and listless. Primary union occurred in the wound, but the tissues seemed "flabby." She was out of bed and sitting in a chair on the twelfth day. Later she began to vomit, developed a tender mass below the liver and a high temperature with chills.

A second operation revealed a few drops of pus beneath the liver and many adhesions of the liver to the pylorus, duodenum and colon. There was no definite collection of pus or bile. Death ensued twenty-four hours later (December 15, 1933), on the seventeenth day after operation.

An autopsy showed an area of necrosis above the gall-bladder fossa buried in the liver substance. There was also a peritonitis in the pelvis with thick patches of grayish fibrin evidently of some days' duration. There was no evidence of any biliary leakage.

Drainage might (?) have saved this patient, but the two other deaths chronicled above, with similar findings at autopsy, were both "drained" cases.

All three patients had been ill a long time before operation, and were suffering from poor nutrition.

Note the ages of three of the four patients who died—fifty-eight, sixty-one and sixty-seven years. Goldish and Gillespie have shown that the average age of the patients who died after operation was 10.3 years greater than that of those who lived.

There was one impressive fact about Cases II, III and IV. Each of them seemed, in an indefinable way, to lack the power of resisting infection.

Two were drained and one was not drained at the time of operation, yet the autopsy findings were almost identical in all three—pelvic abscess.

Severe liver damage was present in all. The question arises, "Does the liver help to prevent infection aside from its digestive and nutritional functions?"

In this series of fifty-five cases, there were no deaths from pulmonary complications, nor were there any "liver deaths," with high temperature, following operation.

The fallacy of statistics in a small series such as this is well shown by the fact that we had only two deaths in our first forty-eight cases—about 4 per cent.—while there were two additional deaths in our next seven cases, making a total of four deaths in fifty-five cases, or about 7.3 per cent. mortality in all.

SURGICAL TREATMENT OF CHOLECYSTITIS

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ACUTE CHOLECYSTITIS*

A STUDY OF 75 PROVEN CASES WITH SUBSIDING OR SUBSIDED CLINICAL
MANIFESTATIONS AT THE TIME OF OPERATION

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THE typical syndrome of acute cholecystitis consisting of abdominal pain, tenderness, muscular rigidity, nausea and vomiting, a palpable mass in the upper abdomen, fever, a rapid pulse rate and leucocytosis, is so well known as hardly to require repetition. In a given case, after a varying period of time, these clinical manifestations undergo alteration, and become either more marked or else commence to subside. If the former occurs, the assumption is that the inflammatory process is progressing. After a period of observation, the length of which depends on the severity of the manifestations and the judgment of the surgeon, if the course is one of progression the patient is usually operated upon. The pathological lesion in the gall-bladder at operation, under such circumstances, is found as a rule to be a severe one. Conversely, if the clinical course is one of improvement, the assumption is that the process is subsiding; and when clinical manifestations become absent, it is assumed that the acute inflammatory reaction has subsided completely.

In our experience these interpretations, although admittedly correct in the majority of instances, are not infrequently erroneous. After having personally encountered several cases of acute cholecystitis of severe grade in the presence of subsiding and subsided clinical manifestations at the time of operation, the author became interested in the subject. As a result, an investigation of the general surgical files of the Mt. Sinai Hospital was made, with the view of determining the nature and frequency of acute inflammatory changes that had persisted in the gall-bladder:

- (1) After clinical manifestations of an acute attack had become minimal.
- (2) After clinical manifestations of an acute attack had disappeared.

During the period from 1929 to 1931, inclusive, 429 operations for gall-bladder disease were performed in the surgical ward services. The clinical diagnoses at the time of admission (*i.e.*, acute, subacute, or chronic cholecystitis) were disregarded and all cases which, on pathological examination, had shown acute inflammatory changes in the gall-bladder, were reviewed. Of these, any case that had had acute or well-marked clinical manifestations at the time of operation was eliminated from further consideration. Seventy-five cases with acute cholecystitis proven by microscopical examination thus remained, of which twenty-three had had only minimal clinical manifestations at the time of operation, while fifty-two had been entirely free of clinical manifestations at a corresponding time.

* Presented before the Surgical Section of the New York Academy of Medicine February 2, 1934.

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A discussion of these cases constitutes the basis of our communication. The reader is reminded that our basis for selection of material for study was a proven diagnosis of acute cholecystitis in each instance. Our presentation is concerned, therefore, with cases in which unusual clinical findings (*i.e.*, mild or absent manifestations) exist in the presence of *proven* acute cholecystitis. It does not deal with the subject of the general pathological findings in *clinical cases* of acute cholecystitis that have subsiding or subsided manifestations. This differentiation, as will be seen later, is of importance.

Each of the diagnoses to be enumerated had been made on microscopical examination, and not on gross inspection at the time of operation. In addition, a gross diagnosis at the time of operation had been made, in the event of such complications as a perforation, fistula between the gall-bladder and a neighboring viscus, pericholecystitic abscess, *etc.*, the presence of which could not have been readily determined in the laboratory after the specimen had been removed from the abdomen. In studying the cases, it was found that gross interpretation of the pathological process in the gall-bladder at the time of operation often had been incorrect, when compared with the later microscopical diagnosis, especially in the advanced cases. For example, in several instances a gross interpretation of gangrenous inflammation proved on microscopical examination to have been merely phlegmonous or hæmorrhagic inflammation with destruction of the mucosa. It was because of such errors that we adopted the criterion of microscopical diagnosis in selecting our cases.

GROUP I.—*Cases of proven acute cholecystitis in the presence of clinical manifestations which have become minimal.*

The following case, although unusual, strikingly illustrates the above syndrome:

A male of fifty-six was admitted to the medical service of the Mt. Sinai Hospital with the following history: Four years previously he had had a one-week episode of attacks of right upper quadrant pain which occurred ten to fifteen minutes after meals. Pain was aggravated by the ingestion of fats or meat. No associated symptoms were present except belching after meals. His physician diagnosed the ailment as due to a gastric ulcer, and prescribed a meat-free and fat-free diet which afforded considerable relief of pain. Belching was relieved by the ingestion of bicarbonate of soda. He felt comparatively well on this régime for four years.

Three weeks before admission, he was seized with sudden, severe pain in the right subcostal region. Pain radiated to the suprapubic region and was accompanied by much pyrosis and belching. Induced vomiting caused some relief of pain, but complete relief was afforded only by morphine. There was no known fever or other associated symptoms. During the three weeks prior to admission he complained only of slight epigastric pain.

On admission, temperature was 100°, pulse 80 and respirations 20. The patient did not appear acutely ill. The only positive abdominal findings were a palpable liver and spleen. There was no rigidity, tenderness or palpable mass present. Blood count: white blood-cells, 12,000; polymorphonuclears, 81 per cent. The admission diagnosis was "penetrating gastric ulcer or carcinoma." He was observed for several days, during which time he complained only of slight epigastric pain. Temperature varied between 100° and 100.6°. Pulse rate, 78 to 92. On the fifth day a barium meal was given and X-ray films taken. On the next day, without warning he was seized with violent pain in the right upper quadrant. Shortly thereafter continuous vomiting began. He

rapidly went into a state of collapse; the abdomen became tender, and board-like rigidity set in. The temperature rose to 104° and the pulse to 116.

At operation, a large amount of blood and pus was noted in the peritoneal cavity, especially along the right side. A large perforated, necrotic gall-bladder filled with stones was found. Cholecystostomy, with drainage of Morrison's pouch, was rapidly performed. The patient did well for two and a half weeks and then developed chills and septic temperature. He died on the twentieth post-operative day. Pus from the gall-bladder was reported to contain *B. coli* and *Streptococcus anhemolyticus*.

At post-mortem examination the essential cause of death was found to be fibrino-purulent pelvic peritonitis.

In the above case, although the clinical manifestations were minimal after the subsidence of an acute episode, an inflammatory lesion of the gall-bladder not only was present but apparently slowly progressed over a period of weeks, until the viscus became almost totally necrotic and ruptured into the free peritoneal cavity. It was not until sudden and extensive peritoneal invasion had occurred that the patient developed alarming manifestations.

The existence of acute inflammatory changes in the gall-bladder associated with clinical manifestations which have subsided to such a degree that they are slight or minimal at the time of operation, has been reported by several authors, including Mentzer,¹ Zininger,² and H. F. Graham.³ In our series there were twenty-three such cases (Table I). They may be divided into two sub-groups, as follows:

(A) Those showing "acute cholecystitis" (unqualified), four cases, or 17.4 per cent.

(B) Those showing more severe acute lesions ranging from acute diffuse inflammation to empyema, gangrene, and perforation, nineteen cases, or 82.6 per cent.

TABLE I

Pathological Diagnoses in 23 Cases of Proven Acute Cholecystitis in the Presence of Minimal Clinical Manifestations at the Time of Operation. (Group I.)

Pathological Diagnosis	Number of Cases
<i>Sub-group A</i>	
Acute inflammation.....	4 cases, 17.4%
<i>Sub-group B</i>	
Acute inflammation with empyema.....	2 cases
Acute inflammation with perforation.....	1 case
Acute inflammation with pericholecystitic abscess.....	1 case
Acute inflammation with perforation and pericholecystitic abscess.....	1 case
Hæmorrhagic inflammation with empyema and pericholecystitic abscess..	1 case
Phlegmonous inflammation.....	1 case
Phlegmonous inflammation with empyema.....	1 case
Suppurative inflammation with empyema.....	1 case
Gangrenous inflammation.....	4 cases
Gangrenous inflammation with empyema.....	3 cases
Gangrenous inflammation with empyema and pericholecystitic abscess...	1 case
Gangrenous inflammation with empyema and perforation.....	1 case
Gangrenous inflammation with perforation, pericholecystitic abscess and cholecysto-gastric fistula.....	1 case
	19 cases, 82.6%

Total number of cases—23

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Examination of Table II reveals the fact that in all of the cases of Group I at the time of operation the temperature was low, ranging from 98.6° to 100°, except in one instance, in which it was 100.4°. The pulse rates varied from 72 to 88 in all but five cases. In these five cases, one was 92, one was 96 and three were 100.

TABLE II

Temperature, pulse rate and residual manifestations in 23 cases of proven acute cholecystitis in the presence of minimal clinical manifestations at the time of operation. (Group I.)

Pathological Diagnosis		Temp.	Pulse	Residual Manifestations
Sub-Group A				
1.	Acute inflammation	100	88	Some rigidity and tenderness in R.U.Q.
2.	" "	98.6	78	Slight R.U.Q. tenderness.
3.	" "	98.6	80	Slight R.U.Q. pain; slight icterus.
4.	" "	98.6	84	Slight R.U.Q. tenderness; small non-tender mass.
Sub-Group B				
5.	Acute " with empyema	99.2	64	Slight R.U.Q. pain.
6.	" " " "	99.0	80	Slight R.U.Q. tenderness.
7.	" " " perforation	99	88	Slight rigidity and tenderness in R.U.Q.
8.	" " " pericholecystic abscess	98.6	76	Slight pain and R.U.Q. tenderness.
9.	" " " perforation and pericholecystic abscess	98.6	88	Slight R.U.Q. rigidity; slight icterus.
10.	Hemorrhagic " empyema and pericholecystic abscess	99.8	84	Moderate abdominal tenderness. Palpable mass.
11.	Phlegmonous inflammation	99.	72	Pain and tenderness in R.U.Q.
12.	" " " with empyema	98.6	80	Slight R.U.Q. tenderness.
13.	Suppurative " " "	99	66	Non-tender mass deep in R.U.Q.
14.	Gangrenous " " "	99.4	76	Tenderness in R.U.Q. Slight icterus.
15.	" " " "	100	86	None
16.	" " " "	99.8	84	Small non-tender R.U.Q. mass. Slight icterus.
17.	" " " "	100	100	None
18.	" " " with empyema	99	86	Slight icterus. Tenderness in R.U.Q.
19.	" " " " " "	100.4	100	Moderate R.U.Q. pain.
20.	" " " " " "	100	100	Pain and tenderness in R.U.Q.
21.	" " " " " and pericholecystic abscess	99.8	96	Non-tender mass in R.U.Q.
22.	" " " with empyema and perforation	100	84	Slight epigastric pain. Rupture into free peritoneal cavity. Then collapse. Pain, tenderness, rigidity in R.U.Q. Vomiting and fever.
23.	" " " with perforation, pericholecystic abscess, and cholecysto-gastric fistula	100	92	Mild R.U.Q. pain. Small hard, irregular, tender mass.

In none of the cases were the manifestations of acute cholecystitis marked at the time of operation. Slight tenderness and rigidity in the right upper quadrant, slight pain, slight icterus and an occasional non-tender or slightly tender mass of variable size, were the usual signs and symptoms present, and it is to be noted that not more than any two of these existed simultaneously in the same patient. Acute clinical manifestations had been replaced by those of minimal severity for from one to twenty-five days prior to the time of

operation. The pre-operative diagnoses included such terms as chronic, subsiding acute, subsided acute and subacute cholecystitis, thus demonstrating the clinical impressions made on the various observers at the time of operation.

Table III lists the number of days that the minimal clinical manifestations had existed prior to the time of operation, and the pathological findings in each case.

TABLE III
Number of Days of Minimal Clinical Manifestations Between Subsidence of Acute Attack and Time of Operation. (Group I.) Pathological Findings in 23 Cases.

1 day	2-4 days	5-7 days	8-21 days	21-25 days
<i>Sub-group A</i>				
Acute..... 1	Acute..... 1	Acute..... 1	0	Acute..... 1
<i>Sub-group B</i>				
Gangrenous with empyema 3	Acute with perforation..... 1	Acute with perforation and pericholecystic abscess.... 1	0	Acute with pericholecystic abscess..... 1
Phlegmonous... 1	Gangrenous... 2	Gangrenous... 1		Gangrenous with empyema and perforation..... 1
	Acute with empyema..... 2	Phlegmonous with empyema 1		
	Gangrenous with empyema and pericholecystic abscess..... 1	Gangrenous with empyema 1		
	Hæmorrhagic with empyema and pericholecystic abscess..... 1	Gangrenous with perforation, pericholecystic abscess, and cholecysto-gastric fistula..... 1		
	Suppurative with empyema 1			
5	9	6	0	3
Total number of cases—23				

GROUP II.—*In addition to the group of cases described above, there is an equally important and perhaps even less recognized group in which acute inflammatory lesions of the gall-bladder exist even after the clinical manifestations have subsided completely.*

As an example, the following illustrative case is briefly presented:

A female of fifty-seven with a history of having had a single attack of acute cholecystitis ten years previously was admitted to the Mt. Sinai Hospital because of cramp-like pain in the right upper quadrant, which radiated to the left shoulder, of forty-eight

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hours' duration. For forty-eight hours the bowels had not moved. During the first thirty-six hours there had been repeated vomiting.

On admission, temperature was 101.4°, pulse rate 104 and respirations 22. The positive abdominal findings were: a palpable liver and a tender, rounded right upper quadrant mass about three inches in diameter. The overlying abdominal wall was rigid, and the abdomen slightly distended. Blood count: white blood-cells, 12,000; polymorphonuclears, 75 per cent. A diagnosis of acute cholecystitis was made. Under conservative treatment all manifestations gradually subsided, and finally disappeared at the end of seven days.

The patient was then kept under observation for an additional period of nine days in order to insure complete subsidence of the inflammatory process before operation. During these nine days she was up and about, had normal temperature and was entirely free of symptoms.

At operation, a tense, acutely inflamed gall-bladder measuring 5 inches by 3 inches was found covered with inflamed adherent omentum. The lumen contained purulent bile and two large stones. Several small abscesses were seen in the wall of the viscus. Cholecystectomy with drainage was performed. Convalescence was uneventful.

Pathological Report.—Chronic and acute suppurative cholecystitis.

In the above case, the patient was entirely symptom-free for a period of nine days before operation, and pulse and temperature were normal. Nevertheless, at operation, acute suppurative cholecystitis was found.

In our series, there were fifty-two cases of proven acute cholecystitis in which complete subsidence of clinical manifestations had occurred prior to the time of operation. (Table IV.) The larger number of cases in the group with absent clinical manifestations as compared with the group with minimal manifestations (*i.e.*, fifty-two as compared with twenty-three) reflected the general tendency of most of the operators to await what they believed to have been complete subsidence of the inflammatory process.

The cases can be divided into two sub-groups, as follows:

(A) Those showing "acute cholecystitis" (unqualified), twenty-nine cases, or 55 plus per cent.

(B) Those showing more severe acute lesions ranging from acute diffuse inflammation to empyema, gangrene, and perforation, twenty-three cases, or 44 plus per cent.

TABLE IV

Pathological Diagnoses in 52 Cases of Proven Acute Cholecystitis in Which Complete Subsidence of Clinical Manifestations Had Occurred Prior to the Time of Operation. (Group II.)

Pathological Diagnosis	Number of Cases
<i>Sub-group A</i>	
Acute inflammation.....	29 cases, 55+%
<i>Sub-group B</i>	
Acute diffuse inflammation.....	2 cases
Acute inflammation with acute pericholecystitis.....	2 cases
Acute inflammation with empyema.....	2 cases
Acute inflammation with empyema and perforation.....	1 case
Acute inflammation with mural abscess.....	3 cases
Hæmorrhagic inflammation.....	4 cases

TABLE IV *Continued*

Ulcerative inflammation.....	2 cases
Ulcerative inflammation with empyema.....	1 case
Phlegmonous inflammation with empyema.....	1 case
Suppurative inflammation with perforation.....	1 case
Suppurative inflammation with pericholecystic abscess.....	1 case
Suppurative inflammation with mural abscess.....	1 case
Suppurative and hæmorrhagic inflammation.....	1 case
Gangrenous inflammation.....	1 case
<hr/>	
23 cases, 44+%	

Total number of cases—52

In none of the cases of Group II were there manifestations, at the time of operation, which are usually associated with acute cholecystitis, such as severe pain, tenderness, rigidity, vomiting, palpable mass or jaundice. While on many of the charts, there were no notes concerning the physical findings immediately preceding operation, it was found that in these the nurse's charts stated that the patients had no subjective complaints. In other words, all patients were free of symptoms referable to their disease, while in addition in many of the cases the physical examinations performed immediately prior to operation were essentially negative. Our later discussion will deal with sub-group A of Group I and sub-group A of Group II as one unit, and with sub-group B of Group I and sub-group B of Group II as a second unit. Any error which may therefore have occurred in the classification of a border-line case is minimized.

Examination of Table V reveals the fact that in forty-three of the fifty-two cases, the temperature was 99° or less. In four it was 99.4°, in three it was 99.6°, and in two it was 99.8°. In forty-three cases, the pulse rates were 84 or less. In four they were 86, in four they were 88, and in one it was 90.

TABLE V

Temperature and Pulse Rate in 52 Cases of Proven Acute Cholecystitis in Which Complete Subsidence of Clinical Manifestations Had Occurred Prior to the Time of Operation. (Group II.)

Pathological Diagnosis	No. of Cases	Temp.	Pulse
<i>Sub-group A</i>			
Acute inflammation.....	25	98.6-99°	23 cases 72-80
	2	99.4	3 cases 84
	1	99.6	2 cases 88
	1	99.8	1 case 90
	<hr/>		
	29		
<i>Sub-group B</i>			
Acute diffuse.....	2	98.6	64
		98.6	80
Acute with acute pericholecystitis.....	2	98.6	72
		98.6	84

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TABLE V *Continued*

Acute with empyema.....	2	98	80
		98.6	76
Acute with empyema and perforation.....	1	98.6	88
Acute with mural abscess.....	3	98.6	86
		98.6	76
		99.0	80
Hæmorrhagic.....	4	98.6	80
		99.6	84
		99.4	78
		99.6	86
Ulcerative.....	2	99.4	86
		99.8	84
Ulcerative with empyema.....	1	98.6	86
Phlegmonous with empyema.....	1	98.6	70
Suppurative with perforation.....	1	99	88
Suppurative with pericholecystitic abscess.....	1	98.6	80
Suppurative with mural abscess.....	1	98.6	80
Suppurative and hæmorrhagic.....	1	99.4	80
Gangrenous.....	1	99	84
	—		
	23		

Total number of cases—52

The acute manifestations had been absent for periods of from one to thirty-five days before operation. Table VI lists the number of days after the subsidence of acute manifestations that operation was performed, and the pathological findings in each case. In one case the patient had been free of acute manifestations since the last attack, three years previously. She was operated upon because of chronic digestive symptoms, after an X-ray of the gall-bladder had revealed the presence of stones. The pre-operative diagnoses included such terms as subsided acute, subsiding acute, and chronic cholecystitis.

General Discussion.—It is to be noted that 44 plus per cent. of the cases in Group II (absent manifestation group) showed advanced lesions on microscopical examination as compared with 82.6 per cent. of the cases in Group I (minimal manifestation group). (See Tables I and IV.) These figures are in keeping with the general concept that the more marked the clinical manifestations, the more apt are advanced lesions to be present at the time of operation. Examination of Tables III and VI reveals that the pathological findings in identical time-groups vary considerably, and that the nature of the residual manifestations (whether mild or absent) gives no clue as to the severity of the lesion found in any given case at operation.

A study of the hospital charts of the twenty-three cases in Group I (minimal manifestation group) led to the classification of twelve as mild or moderately severe, while eleven could be classified as very severe at the height of the attack. Of the fifty-two cases in Group II (absent manifestation group), sixteen were classified as mild and thirty-six as severe at the height of the attack. It was noted that the lesions found on microscopical

TABLE VI
Number of Days of Absent Clinical Manifestations Between Subsidence of Acute Attack and Time of Operation. (Group II.) Pathological Findings in 52 Cases.

1 day	2-4 days	5-7 days	8-10 days	11-14 days	15-21 days	21 days or longer
Acute..... 1	Acute..... 11	Acute..... 3	Acute..... 4	Acute..... 2	Acute..... 4	Acute..... 4
<i>Sub-group A</i>						
<i>Sub-group B</i>						
Haemorrhagic... 1	Acute diffuse... 1	Acute diffuse... 1	Acute with mural abscess.... 2	Haemorrhagic... 1	Acute with mural abscess.... 1	Ulcerative with empyema..... 1
	Acute with empyema..... 2	Suppurative with mural abscess..... 1				Suppurative with pericholecystitic abscess..... 1
Ulcerative..... 1	Haemorrhagic... 2					Acute with empyema and perforation..... 1
Acute with pericholecystitis... 2	Phlegmonous with empyema. 1					Longest interval three years.
Suppurative and haemorrhagic... 1	Suppurative with perforation..... 1					
Gangrenous.... 1	Ulcerative..... 1					
2	17	11	7	3	5	7
Total number of cases—52						

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examination varied greatly and bore no consistent relationship to the severity of the clinical manifestations at the height of the attack. In other words, patients in whom the attack at its height could be classified as mild or moderately severe showed lesions ranging from acute inflammation (unqualified) to gangrene with perforation, while patients in whom the attack could be classified as very severe showed the same variations in the extent and severity of their lesions.

Consideration is now given the four cases with minimal manifestations and the twenty-nine cases with absent manifestations at the time of operation, that showed "acute inflammation" (unqualified). The former are of no unusual significance, and the minimal clinical manifestations at the time of operation are entirely compatible with the operative findings. These are the cases which one would properly classify as subsiding. If not actually subsiding at the time of operation they are assuredly capable of subsiding, since the pathological changes are not advanced. The remaining twenty-nine cases are of interest because they demonstrate that acute changes may exist in the gall-bladder of a patient who at the time of operation is free of subjective complaints and clinical manifestations. Since the manifestations were more severe at some previous time, it is fair to assume that in these cases the inflammatory lesions in all probability were also subsiding. Considering for a moment the entire thirty-three cases it is to be noted that the pathological lesions found in the absent-manifestation and subsiding-manifestation groups were identical, *i.e.*, "acute inflammation" (unqualified). Why some of the patients should exhibit minimal manifestations and others be free of manifestations in the presence of identical pathological lesions cannot be answered. Why this general group should have absent or minimal manifestations, as compared with the usual variety which exhibits severe manifestations in the presence of identical pathological findings, is also inexplicable.

There are now to be considered the nineteen cases with minimal manifestations and the twenty-three cases with absent manifestations at the time of operation that showed severe pathological changes, *i.e.*, hæmorrhagic, phlegmonous, suppurative, and gangrenous inflammation, empyema, perforation and pericholecystitic abscess. The former (nineteen cases) are of considerable interest since they demonstrate that advanced inflammatory lesions may and do exist in the presence of manifestations that became minimal prior to the time of operation, after being marked at the height of an attack. The remaining twenty-three cases are outstanding, in so far as the operative and pathological findings paralleled those of the aforementioned nineteen, while the clinical manifestations were not only less, but absent. Both of the groups re-emphasize the fact that there are exceptions to the rule that the severity of the clinical manifestations is an indication of the severity of the pathological process. *In other words, advanced grades of inflammation may exist in the complete absence of clinical signs and symptoms at the time of operation, or the presence of minimal clinical signs and symptoms at the time of operation.*

It is impossible to state with certainty whether the process in any given case of these forty-two was subsiding or progressive at the time of operation. Whether or not subsidence would have occurred likewise cannot be stated, as operation ended the opportunity for further observation. In spite of the marked pathological changes present, however, one may assume that most of the cases were at least capable of subsiding.

In eleven of the forty-two cases, the diagnosis of "gangrenous cholecystitis" was made on pathological examination. In each of the cases, the gangrenous process had diffusely involved the gall-bladder wall. When gross gangrene supervenes, the pathological process may be considered to be irreversible and to definitely preclude the possibility of subsidence. Progression of the process in the eleven cases was therefore the only possibility. An analysis of our figures shows that of the cases with advanced lesions, gangrenous cholecystitis was reported in ten (52.6 per cent.) of the group of nineteen that had minimal manifestations, as compared with only one (4.3 per cent.) of the group of twenty-three that had absent manifestations.

In addition four cases without gangrene, of the forty-two showing advanced lesions, were associated with pericholecystitic abscesses of large size. Three occurred in the minimal-manifestation group, and one in the absent-manifestation group. These large pericholecystitic abscesses often do not absorb, especially if stones are present. Instead they become walled-off by dense organized exudate and may persist for long periods of time, unless free drainage occurs through the establishment of a communication back into the gall-bladder in the presence of a patent cystic duct. Drainage may also occasionally occur as the result of perforation into an adjacent hollow viscus such as the duodenum, stomach, or occasionally the colon. Small pericholecystitic abscesses may of course absorb spontaneously, as do abscesses of small size elsewhere. An abscess of fair size therefore constitutes a lesion which is usually persistent and at the same time locally active, rather than one which is subsiding. These lesions at some later time may occasionally disappear as the result of spontaneous drainage as described above, or may become progressive if a flare-up of the low-grade infection occurs.

Zininger² states that "when empyema of the gall-bladder once develops it is unlikely to subside spontaneously, and may progress to gangrene and rupture of the wall." Although this statement may be true, we have purposely refrained from classifying as progressive, nine cases in this series that had empyema without actual gangrene of the gall-bladder wall. In short, regardless of the potentiality of a case to become progressive we have purposefully designated as progressive lesions only those that were actually irreversible at the time of operation. We therefore have considered only the eleven cases with gangrene and the four cases with good-sized pericholecystitic abscesses to have had non-subsiding lesions. A study of these fifteen cases reveals that thirteen had minimal clinical manifestations, while only two had absent manifestations. The thirteen cases constitute 68.4 per cent.

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of the former group, while the two cases constitute only 8.7 per cent. of the latter group.

The fifteen cases represent, also, 20 per cent. of the entire group of seventy-five under consideration. It is to be re-emphasized that this statement does not mean that 20 per cent. of all cases of apparently subsiding or subsided acute cholecystitis have progressive lesions. It means that of a series of seventy-five cases of *proven acute cholecystitis in the presence of minimal or absent clinical manifestations*, 20 per cent., had progressive lesions, while the remaining 80 per cent. had lesions that were subsiding or were considered capable of subsidence.

SUMMARY

(1) Four hundred twenty-nine operated cases of cholecystitis were reviewed. Of these, a special group of seventy-five with acute cholecystitis proven by pathological examination, that were found to have had minimal or absent clinical manifestations at the time of operation, were studied.

(2) Of twenty-three cases that had minimal clinical manifestations at the time of operation:

(a) 17.4 per cent. showed "acute cholecystitis" on pathological examination, and

(b) 82.6 per cent. showed more advanced acute lesions.

(3) Of fifty-two cases without clinical manifestations at the time of operation:

(a) 55 plus per cent. showed "acute cholecystitis" on pathological examination, and

(b) 44 plus per cent. showed more advanced acute lesions.

(4) Clinical manifestations were minimal or absent for from one to thirty-five days prior to the time of operation. In one case the patient was free of acute symptoms for three years preceding operation.

(5) The percentage of advanced pathological lesions was 82.6 in the group that had minimal clinical manifestations at the time of operation, as compared with 44 plus in the group with absent manifestations at the time of operation.

(6) The percentage of progressive pathological lesions was 68.4 in the group that had minimal clinical manifestations at the time of operation, as compared with 8.7 in the group with absent manifestations at the time of operation.

(7) In any given case, the severity of the attack bore no consistent relationship to the severity of the lesion found in the gall-bladder at operation.

(8) In any given case, the duration and nature of the residual manifestations, if any, bore no consistent relationship to the severity of the lesion found in the gall-bladder at operation.

(9) Twenty per cent. of the cases in the entire series were considered to have progressive lesions, while 80 per cent. were apparently either going on to subsidence or were considered capable of subsidence.

On the basis of this study, certain conclusions are warranted in regard to the clinical management and indications for operation *in cases of acute cholecystitis in which the clinical manifestations have subsided or are subsiding*. We may in general conclude that if the signs and symptoms are subsiding uninterruptedly, fairly promptly, and completely, there is no indication for *urgent* surgical intervention. On the other hand, because of the possibility (even though slight) of the existence of an advanced lesion, we feel that these patients should be operated upon within a short period of time after subsidence has occurred. Since nothing is to be gained by delay and in some cases much may be lost if perchance a progressive lesion is present, it has been the practice of the members of this service to operate within several days of subsidence, if in general no contra-indications to surgical therapy exist.

On the other hand, once subsidence has begun, if clinical manifestations persist even though they be slight, we feel that fairly prompt operation is indicated. Muscular rigidity, spontaneous pain, tenderness, a palpable mass, and fever if recrudescient or persistent even though not marked, are considered to be of great significance. Furthermore, the presence of an irregular tender mass even of small size strongly suggests the possibility of a pericholecystic abscess. White blood-cell and differential counts when high are confirmatory, but when only slightly elevated or normal do not militate against the diagnosis of a progressive lesion. After the first phase of infection has subsided and clinical manifestations have become slight or absent, blood counts often are apt to be unreliable as indicators of the severity and extent of the inflammatory lesion present. We therefore prefer to place our reliance in the later phases of infection on clinical signs and symptoms in deciding on indications for surgical intervention.

Since adopting the plan of treatment described above, we have on not a few occasions encountered advanced and progressive lesions in the presence of subsiding and subsided clinical manifestations. Although our series is comparatively small at present, the results thus far obtained have warranted the continuation of treatment along the lines we have outlined.

CONCLUSIONS

- (1) Acute inflammatory changes may exist in the gall-bladder of a patient with minimal or absent clinical manifestations at the time of operation.
- (2) The pathological changes range from simple acute inflammation to hæmorrhagic, phlegmonous, suppurative, and gangrenous inflammation, empyema, perforation and pericholecystic abscess.
- (3) In general, the patients with minimal manifestations at the time of operation show a considerably higher percentage of advanced and progressive lesions than the patients with absent manifestations at the time of operation.
- (4) Eighty per cent. of the lesions in a selected series of seventy-five cases were considered to be subsiding or capable of subsidence. The remaining 20 per cent. were considered to be progressive in nature.

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(5) In any given case, it is impossible to determine the exact nature and extent of the inflammatory lesion before operation.

(6) In cases of acute cholecystitis, if subsidence once begun does not proceed uninterruptedly, fairly promptly, and completely, early operation is indicated.

(7) In cases of acute cholecystitis with subsided clinical manifestations, operation, early rather than late in the "interval," is indicated.

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PERFORATION OF THE GALL-BLADDER *

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PERFORATION of the gall-bladder is considered in surgical text-books to be a rather rare complication of biliary disease. That acutely inflamed gall-bladders do not perforate more frequently has been attributed to the excellent blood supply of the viscus, its tough fibromuscular coat and the action of the bile in reducing the virulence of the pathogenic organisms in the gall-bladder. On Surgical Service C of the Hospital of the University of Pennsylvania this complication has been encountered nine times in the past eleven years in 490 consecutive biliary admissions. The occurrence of this series of perforations led to a review of the subject and an analysis of our cases.

Karullon,¹ in a series of 6,114 consecutive autopsies, found gall-stones present in 572, three of these cases having succumbed to a perforation of the gall-bladder.

The following table shows the incidence of perforation of the gall-bladder in several large series of biliary cases reported by various authors. (Table I.)

From the accompanying table it is evident that perforation of the gall-bladder occurs in from 1 to 3 per cent. of all cases of biliary disease. The incidence of this complication has not been generally appreciated in the past and warrants more careful consideration by all surgeons.

TABLE I

Incidence—Perforation of the Gall-Bladder

Author	Cases	Perforated		Operation		Mortality
		Gall-Bladder	Calculi	ectomy	ostomy	
Gosset.....	—	111	33%	—	—	52%
Georg.....	—	348	—	—	—	42%
Fifield.....	1,066	27 (2.5%)	26 (96%)	5	22	44%
Fisher and Mensung..	300	4 (1.3%)	—	—	—	—
McWilliams.....	3,180	29 (.9%)	—	—	—	—
Alexander.....	1,000	20 (2.0%)	12 (60%)	6	14	35%
Mitchell.....	1,270	16 (1.2%)	11 (69%)	10	5	43%
Eliason and McLaughlin.....	500	9 (1.8%)	8 (89%)	1	8	11%

Although calculous cholecystitis is encountered in the female patient many times more frequently than in the male, perforation of the gall-bladder occurs in a higher relative proportion of male cases. In our series of nine cases, 44 per cent. were males.

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Perforation of the gall-bladder has been reported in patients ranging in age from early youth to eighty years. Power and Johnston⁵ reported the case of a child two and a half years of age with a perforated empyema of the gall-bladder secondary to an *ascaris lumbricoides* infection of the biliary tract. Most of the reported cases occurred in patients between fifty and sixty-five, the average of our group being 57.3 years.

In the majority of cases of perforated gall-bladder disease a definite history of previous attacks of cholecystitis may be elicited. Years ago Grant⁶ called attention to the importance of long-standing biliary disease as a predisposing factor in perforation of the gall-bladder. Alexander³ elicited a definite history of gall-stone colic in fifteen of his sixteen cases and Georg⁷ reported similar findings in twelve of his twenty cases. Gosset, *et al.*⁸ found a history of gall-stone disease in 33 per cent. of their collected series of 111 cases. In our series of nine cases, all but one had a history of previous acute attacks of cholecystitis varying in number from one to eight. A review of these cases revealed a history of biliary disease extending back from eighteen months to fifteen years, the average being six years before admission with a perforation. In four cases there was a history of jaundice with previous acute attacks.

Six of our nine cases were admitted directly to the surgical service with provisional diagnoses of acute surgical abdomens. The shortest duration of the present illness on admission was four days, the longest was ten months. With the exception of this one case in which the history went back for a period of months the average duration of the present illness was twenty days.

Severe right upper quadrant pain was the predominant symptom in five cases. In two of these, agonizing pain was also present in the epigastrium. In one case the pain was not relieved by $2\frac{1}{2}$ grains of morphine in an eighteen-hour period. Three cases complained of only moderate right upper quadrant pain on admission, while one had no pain. There was radiation of the pain to the right scapula in three cases. Persistent nausea and vomiting were prominent features in six of the nine patients while the remaining three gave a history of gaseous distention and anorexia.

TABLE II
Symptomatology—Perforation of the Gall-Bladder

Case	Previous History	Duration P. I.	Pain	Radiation of Pain	Nausea Vomiting	Chills	Icterus
1	5 yrs.	42 da.	sev.	no	+	yes	—
2	1 mo.	30 da.	no	no	+	no	—
3	25 yrs.	4 da.	mod.	no	o	no	+
4	18 mo.	8 da.	sev.	yes	+	no	+
5	15 yrs.	4 da.	sev.	no	+	no	—
6	5 yrs.	30 da.	mod.	no	+	no	+
7	8 yrs.	10 mo.	mod.	no	+	yes	+
8	9 yrs.	8 wks.	sev.	yes	+	no	+
9	5 yrs.	8 wks.	mod.	no	+	no	o

It is evident that very severe right upper quadrant pain with or without shoulder radiation, accompanied by persistent nausea and vomiting which does not promptly respond to palliative therapy should lead one to suspect acute disease (calculus) of the gall-bladder.

The essential physical findings in this series of cases are listed in Table III.

TABLE III
Physical Findings—Perforation of the Gall-Bladder

Case	Local Tenderness	Palpable Mass	Temperature	Pulse	W. B. C.
1	+	+	101	110	11,700
2	+	+	99	100	10,200
3	+	—	98	70	6,400
4	+	—	101	120	21,000
5	+	—	102	110	12,500
6	+	+	101	120	18,000
7	+	+	98	88	9,000
8	+	+	98	100	5,600
9	+	+	99	110

On admission a definite palpable mass was found in six cases, while two of the remaining cases were so exquisitely tender that satisfactory palpation of the abdomen could not be carried out. None of the patients had the diffuse abdominal tenderness and rigidity commonly associated with a widespread or generalized peritonitis.

Röntgenological studies were carried out in five patients. Two were subjected to a gastro-intestinal series because of persistent vomiting. In one of these cases visible peristalsis had been observed on clinical examination. In both cases an X-ray diagnosis of pyloric obstruction was made, being attributed in one to gastric carcinoma. Although the same diagnosis was considered in the second case, the röntgenologist suggested a diagnosis of empyema of the gall-bladder with secondary gastric retention as a possibility. Graham-Cole tests were carried out in three cases, the gall-bladder failing to visualize in each instance. In two of these cases X-ray of the chest disclosed the presence of a fixed right diaphragm with haziness at the base of the right lung. This was interpreted as being due to fluid in the pleural cavity secondary to the subdiaphragmatic lesion.

A correct pre-operative diagnosis of a perforated gall-bladder is quite unusual as shown by a review of the reported cases in the literature. In those cases in which there is a widespread soiling of the peritoneum without localization of the lesion, diagnoses of perforated ulcer, ruptured appendix, and diffuse peritonitis of undetermined origin have been frequently made. In cases which remain localized, acute gall-bladder disease, empyema of the gall-bladder, pyloric obstruction, intestinal obstruction, and abdominal tumor of unknown origin are the most commonly listed diagnoses. Mitchell⁴ reports that before operation the correct diagnosis was made in but one of his series of sixteen cases. Georg⁷ states that none of his twenty cases

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were correctly diagnosed before operation although the gall-bladder was considered to be the diseased organ in ten. In our series of nine cases the pre-operative diagnoses were acute perforated cholecystitis, one; acute calculous cholecystitis, seven; pyloric obstruction, one. Associated pre-operative diagnoses were acute pancreatitis, one; stone in the common duct, two; and empyema of the gall-bladder, four.

A correct pre-operative diagnosis of perforation of the gall-bladder is not often made because such a diagnosis is seldom considered as one of the possibilities. As mentioned above, the teaching for so many years has been to the effect that acute gall-bladder disease rarely undergoes perforation, that one is apt to omit this possibility in making a differential diagnosis.

The essential operative findings in our series of cases are listed in Table IV.

TABLE IV
Operative Findings—Perforation of the Gall-Bladder

Case	Localized	Stones		Culture	Operation		Result
	Abscess	Cystic	Extra Cystic		ectomy	ostomy Drainage of Abscess	
1	yes	yes	yes	strep.	—	yes	good
2	yes	yes	yes	sterile	—	yes	good
3	yes	yes	yes	no pus	yes	—	good
4	yes	yes	no	sterile	—	yes	good
5	yes	yes	yes	B. a. lacti.	—	yes	good
6	yes	yes	yes	B. a. lacti.	—	yes	good
7	none	no	yes	no pus	—	yes	good
8	yes	yes	no	sterile	—	yes	died
9	yes	no	no	staph. colon	—	yes	good

In seven of our nine cases a localized abscess was present at operation, secondary to a perforated gall-bladder.

In this group of cases the most common site of perforation was found to be in the fundus of the gall-bladder. Only one case was encountered in which the gall-bladder had been partially successful in perforating directly into the intestinal tract. In this patient a circular gall-stone two centimetres in diameter had passed through the gall-bladder wall and was lodged within the meso of the hepatic colon without entering the lumen of this viscus. None of the cases observed had developed intestinal obstruction due to gall-stones. Mitchell⁴ states that when this complication occurs, 65 per cent. of the obstructions are found in the ileum, 21 per cent. in the duodenum and jejunum, 10 per cent. at the ileo-cæcal valve and the remaining cases either in the sigmoid colon or at the pylorus. Bennett⁹ in a review of 3,064 collected cases of small bowel obstruction found only twenty-eight cases due to gall-stones and concluded that this was a rather rare complication.

As has been frequently stated, the essential requirements for the development of an empyema of the gall-bladder are the presence of pathogenic

organisms and an obstruction of the cystic duct, usually due to stone. The operative findings in this series of cases reaffirm this statement. In seven patients stones were present in the gall-bladder at operation and with but three exceptions were also found outside the gall-bladder. Alexander³ reports the presence of stones in 60 per cent. of his twenty cases and Fifield² found calculi in 93 per cent. of his group of twenty-eight cases. That a gall-bladder may become gangrenous and perforate without presence of stones, as a result of a thrombosis of the vessels supplying its walls, is borne out by numerous reports in the literature. Tongs¹⁰ has recently reported such a case in which the cystic duct was completely occluded by debris at operation, and although the gall-bladder was gangrenous, no stones were present.

In our cases the amount of pus removed from the abscess cavities varied from 60 cubic centimetres to 1,000 cubic centimetres. That extremely large abscesses may develop in association with a perforation of the gall-bladder is evidenced by Overholt's¹¹ report of one from which over 4,000 cubic centimetres of pus were removed at operation.

Cultures of the pus in seven of our cases did not disclose the presence of virulent organisms except in two instances. While the colon bacillus, streptococcus and staphylococcus are the usual organisms responsible for acute cholecystitis, it is well recognized that they are primarily localized within the gall-bladder walls and one might well expect to find the cultures of pus obtained at operation sterile, as is frequently the case.

Cholecystostomy with drainage of the abscess cavity was the operative procedure carried out in seven of the nine cases. In the other two cases, in which the old healed perforations of the gall-bladder were evidenced by the presence of encysted gall-stones in the pericholecystic fat in one and in the meso of the hepatic colon in the other, choledochostomy for associated common duct calculi was performed in each and a cholecystectomy in one.

CASE I.—J. S., male, aged sixty, was admitted to the University Hospital complaining of recurrent attacks of upper abdominal pain, associated with jaundice. Two and one-half years before admission the patient had his first attack of epigastric pain associated with anorexia, distention and jaundice. He was then free from symptoms until four months prior to admission when a similar attack was experienced. Subsequently the attacks recurred at intervals of a month, each characterized by epigastric pain, distention and jaundice. On admission, the temperature, pulse and respiration were normal. The patient was well developed and nourished, there was no evidence of weight loss but definite icterus of the sclera and skin was noted. There was a slight tenderness deep in the right upper quadrant. No masses were palpable. *Diagnosis.*—Calculous obstruction of the common duct. At operation a dense mass of adhesions was found in the right upper quadrant, involving all the structures in this region. Scattered over and adherent to the peritoneal surface of the duodenum, hepatic flexure, and the omentum in this area were a dozen or more small gall-stones of variable sizes. The gall-bladder, which was subacutely diseased, was removed. The common duct was opened and several calculi removed, after which a T-tube drain was installed. Two of the gall-stones were removed from the free peritoneal surface and sent to the laboratory for confirma-

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tion of their suspected nature. They were reported as gall-stones. The patient's convalescence was entirely uneventful and at the time of discharge from the hospital his wound was healed.

CASE II.—R. J., male, aged fifty-seven, was admitted to the hospital complaining of upper right quadrant pain. For seven years he had been suffering with upper right quadrant pain associated with jaundice and vomiting. He had lost seventy pounds in weight during the last year. On admission the temperature, pulse, and respiration were normal. Examination showed a definitely jaundiced man with evidence of weight loss. There was slight tenderness with resistance in the right upper quadrant. No definite masses were palpable. White blood cells, 9,000; urine, negative, Wassermann, negative. Icteric index was 40. Graham-Cole test showed a non-functional gall-bladder. *Diagnosis.*—Calculous obstruction of the common duct. At operation the colon and duodenum were found to be intimately adherent in the region of the gall-bladder. No gall-bladder could be definitely found, the structure having been evidently completely obliterated. One faceted stone the size of a hazelnut was removed from the common duct and a T-tube placed in this duct. Lodged in the meso of the hepatic flexure of the colon adjacent to the gall-bladder fossa, a circular, firm gall-stone 2.5 centimetres in diameter was found. It had not eroded into the lumen of the colon and was removed without opening this structure. Routine closure with drainage was done.

In this group of nine cases there was only one death. This patient succumbed twenty-four hours after operation from acute cardiac failure. The average number of hospital days was thirty-two. Six of the series have been entirely relieved since operation. One case was left with a persistent mucous fistula while a second returned some months after his first operation with a calculous common-duct obstruction which was successfully relieved at operation.

In the literature, opinions vary in regard to the correct way of handling of acute perforated gall-bladder when such a lesion is found at operation. Georg⁷ advises cholecystectomy as the operative procedure of choice and quotes Mayo and Courvoisier as concurring in this opinion. Fisher and Mensung¹² also advocate cholecystectomy in every case in which this procedure can possibly be carried out. They report the case of a fifty-three-year-old male upon whom they operated twice within a period of three months, a perforated gall-bladder being found each time. Cholecystectomy was performed the second time and the patient recovered.

The mortality in all the reported series is high and leaves much to be desired. Georg⁷ in a collected series of 348 cases found the mortality to be 42 per cent. Mitchell⁴ reports a mortality of 43 per cent., Gosset, *et al.*,⁸ 52.5 per cent., Alexander,³ 58.3 per cent. and Fifield,² 42.4 per cent. In this series of nine cases the mortality was 11 per cent. Judd and Phillips¹⁴ report a series of sixty-one cases of perforation of the gall-bladder. In all but two the peritonitis was definitely localized. Cholecystectomy was performed in forty-eight of the sixty-one, with five deaths, and cholecystostomy was done in thirteen with one death.

Obviously the conditions found at operation would influence the type of surgical procedure that could be carried out in a given case. In this series a definite localized abscess surrounding the gall-bladder was present

in seven cases. In these, drainage of the abscess cavity with cholecystostomy was the only procedure which could possibly have been undertaken from a technical standpoint and with safety to the patient. In the other two cases in which the perforations had healed at the time of operation, a cholecystectomy was carried out in one case while the common duct was drained in both. In this series no cases with widespread dissemination of bile through the peritoneal cavity or diffuse peritonitis were encountered. This probably accounts for the fact that in the entire series there was only one fatality.

Realizing that perforation of the gall-bladder is not the rare catastrophe that it was once considered to be, more emphasis may justly be placed upon the necessity of urging all patients with attacks of recurrent calculous cholecystitis to submit to cholecystectomy while their gall-bladder disease is quiescent. Much may also be accomplished toward lowering the high mortality in cases that have already perforated by arriving more promptly at a correct diagnosis. Delay on the part of both the patient and the attending physician probably is responsible for the loss of many patients with this complication each year. On the assumption that the severe biliary symptoms represent only another attack of acute cholecystitis which will subside as the preceding ones have done, these cases are permitted to progress beyond the point where operation can save them. The difficulty of ascertaining the extent of the pathological process affecting an acute gall-bladder by clinical examination has been recently emphasized by Mentzer.¹³ If all cases of severe recurrent cholecystitis which do not promptly subside under adequate palliative therapy within a reasonable time are considered as potential perforations of the gall-bladder, this high mortality will be materially reduced.

SUMMARY AND CONCLUSIONS

- (1) Perforation of the gall-bladder occurs in from 1 to 3 per cent. of all cases of biliary disease.
- (2) Perforation usually occurs in patients who have had a long-standing history of chronic calculous cholecystitis.
- (3) A correct diagnosis is rarely made before the patient is subjected to diagnostic laparotomy.
- (4) Early operation is urged in all cases of acute cholecystitis which do not promptly subside under adequate palliative treatment.
- (5) The operative procedure in each case is entirely dependent upon the nature of the lesion found at operation.
- (6) The mortality of perforated gall-bladder disease is extremely high, ranging from 10 to 58 per cent.
- (7) Unless there is a definite contra-indication, all cases with recurrent attacks of chronic calculous cholecystitis should be urged to submit to surgery during a quiescent period.

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ACUTE FREE PERFORATION OF THE GALL-BLADDER

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THE prevailing conservative attitude on the part of surgeons in the treatment of acute diseases of the gall-bladder, as contrasted with that of early intervention in acute appendicitis, is based on the assumption that the great majority of the former will subside.

The occurrence of a recent case of acute free perforation of the gall-bladder in my practice has led me to investigate this subject with results which I believe are of interest. I append below the history of this case of acute free perforation of the gall-bladder with operation and recovery.

CASE REPORT.—Mr. C., aged fifty-seven years. *Previous Illness.*—Eighteen years ago he began having attacks of colicky pain in the right hypochondrium; another attack occurred three years ago accompanied by jaundice, with which he was in bed about a week.

Practical Illness.—On September 16, 1933, developed severe pain in the right upper quadrant which radiated to the back and was followed by vomiting. This was diagnosed as biliary colic and morphia was given. When first seen by me on September 17, 1933, there was a tinge of icterus present. The temperature was 99.2°, pulse 80, and there was moderate tenderness in the right hypochondrium. On September 18, 1933, the pain became more severe, temperature rose to 101° and tenderness was more marked and diffuse with some muscular rigidity, suggesting a definite acute cholecystitis with some localized peritonitis. He was sent to the hospital and carried along on intravenous glucose in saline with nothing by mouth, in the hope that the condition would subside. His white blood cells on September 19, 1933, were 13,100 (polymorphonuclears 75 per cent.) and his condition seemed to be improving, the pain and tenderness became somewhat less marked, and white blood cells on September 20, 1933, were 12,300. On September 20, 1933, about 5 P.M., he suddenly developed an agonizing pain accompanied by signs of shock with cold, clammy sweat; morphia gr. ½ was necessary to control the pain and an intravenous of glucose in saline was given for shock. Generalized abdominal rigidity was evident by the next morning and, as shock had subsided, laparotomy was proceeded with on the diagnosis of perforation of the gall-bladder.

Operation.—On opening the peritoneum free bile was found in large quantities and the gall-bladder was found collapsed and entirely free of any protective adhesions. There was a stone in Hartmann's pouch with a perforation near the neck of the organ. Cholecystostomy was done with removal of a cholesterol stone from the gall-bladder and two cigarette drains placed in Morrison's pouch.

Post-operative.—There was considerable distention for a few days but this was controlled with pitressin and flatus was being expelled freely on the third day, fluids by mouth then being allowed gradually. His fistula persisted, and stools remained clay-colored and some obstruction of the common duct was considered probable. No exploration of the common duct was made at the time of the first operation as his condition precluded anything of this kind.

Second Operation.—On December 7, 1933, the abdomen was again opened and the gall-bladder found to contain bile which could be expressed. The common duct was markedly dilated and a stone could be palpated just above the duodenum. The duct was opened and a soft, crumbly stone, the size of a hazelnut, removed and a T drain

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inserted, and the duct closed about this. The gall-bladder was not removed as it was thought that it might be of future use in establishing a cholecystogastrostomy later, if stricture of the duct should occur. The biliary fistula immediately closed on removal of the drain and the patient has been well and free from jaundice ever since.

Perforations of the gall-bladder may be subdivided into three groups:

(1) Chronic perforations with the presence of a fistulous communication between the gall-bladder and some other viscus.

(2) Subacute perforations where the perforated gall-bladder is surrounded by an abscess walled off by adhesions from the general peritoneal cavity.

(3) Acute perforation of the gall-bladder into the free peritoneal cavity without protective adhesions, as illustrated by the case reported above.

The literature would indicate that acute perforation of the gall-bladder is extremely rare and that the mortality is very high.

Mitchell¹ reports sixteen cases, of which six were acute, in 1,270 gall-bladder operations, giving an incidence of 1.2 per cent. for all varieties with a mortality of 50 per cent.

Alexander² cites 1,000 cases of biliary disease with a somewhat higher incidence of twenty cases, or 2 per cent., twelve being of the subacute variety, and eight or, .8 per cent., being acute free perforations. The mortality in the subacute cases was 25 per cent. and in the acute free perforations was 50 per cent.

Judd³ in a recent article on this subject, reports sixty-one cases of perforation of the gall-bladder, fifty-nine being of the subacute type with walling-off adhesions and two being acute free perforations with a mortality of 50 per cent. Judd states that in seven of these cases of perforation there was a fistulous communication with some other viscus. He also mentions three fatal cases of acute free perforation who were too ill for operation, constituting a total of five cases of this type in all. In a personal communication Judd was kind enough to inform me that these statistics were based on a series of 9,446 gall-bladder operations, giving an incidence of acute free perforations of about .05 per cent. Judd stressed the fact that many of their cases come from a distance and the cases of acute free perforations being too ill to travel are operated on at home. Consequently he felt that his series might not give a true picture of the incidence of this condition.

I undertook to investigate the gall-bladder cases in the Hamilton General Hospital during the last three years and wish to acknowledge my indebtedness to Dr. R. E. Nicholson for the statistical information recorded below.

In a review of 349 operations on the biliary tract at the Hamilton (Ont.) General Hospital we found eight cases of perforation of the gall-bladder classified as follows: Chronic perforation, two cases—one into duodenum, one into colon; subacute perforation, four cases; acute free perforation, two cases.

There were no deaths in this series. This gives a percentage of acute free perforation of .57 per cent. This is ten times the frequency reported by Judd.

CONCLUSIONS.—The conclusions to be drawn regarding acute free perforation of the gall-bladder, I believe, are the following:

(1) This condition occurs comparatively rarely.

(2) It is of sufficient frequency, however, to demand eternal vigilance in the delayed treatment of acute cholecystitis.

(3) The mortality is extremely high as usually reported, but it would appear from our series that prompt recognition and treatment might lower this considerably.

While we realize that our series is comparatively small, it is representative of the work of the average general hospital. I hope that this will stimulate others working under similar conditions to compile statistics so that from these combined figures we may evaluate this condition in its true light.

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ACUTE INFLAMMATION OF THE GALL-BLADDER; CONSERVATIVE OPERATIVE TREATMENT

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TO BEGIN this paper on the morbidity and mortality of the subject under consideration is most uncommon. Let me state at the outset that the success of the ultimate outcome of a patient operated on depends largely on the judgment of the surgeon; this judgment is gained from experience. To a marked degree this is amplified in the treatment of acute inflammations of the gall-bladder. On account of the somewhat contradictory opinions reported and expressed at the last meeting of the American Surgical Association it seems fitting to review our work and tell why our mortality and morbidity have been less than with those advocating radical measures in the treatment of acute cholecystitis. In over twenty-seven months I⁴ have not had a death from the operation of cholecystectomy. This excludes all malignant conditions, and operations on the common duct for cancer of the head of the pancreas. But it does include all types of inflammatory conditions of the gall-bladder with and without adhesions, and the various types of congenital bands. The morbidity of cases operated on in the acute stage is far greater because two operations are usually necessary, whereas if our treatment is followed one operation suffices usually without drainage of the field of operation, about which more will be said later. It has been my experience to witness death within twenty-four or forty-eight hours after operation on cases of acute empyema of the gall-bladder. Whether this is due to the release of a massive infection, or the opening of spaces for the entrance of infection, or a chemical death, has never been settled to my satisfaction. Cultures have not helped much because they are usually sterile. I take exception to the opinions expressed by Stone and Owings,¹ Walton, Kirschner, *et al.*; namely, that acute gall-bladder conditions should be operated upon as an emergency procedure. The tendency of this teaching will result in a far greater mortality than we have observed for the past few years. It will be an annihilating influence, especially if practiced universally by those not able to cope with the increased mechanical difficulties encountered at operation on acute cases. A mortality of 10 per cent. was always the greatest argument used against operation by those treating the diseased gall-bladder medically. Each year our mortality has been reduced because conservative methods have been used by not subjecting the patient to immediate operation. I fear again for a higher mortality because of the wide circulation these papers will receive. A surgeon who operates immediately upon the inception of symptoms of empyema of the gall-bladder is only playing with death; if, however, the surgeon waits two, three, or four days or longer, then he is not taking the risks

of an immediate operation. The proper time for operation may vary with each case, some subsiding in a day or two, when it may be safe to operate, while in others the operation may have to be delayed for a week or ten days on account of the acuity of symptoms. The time for operation is gauged by the physical signs imparted to one's fingers on palpation of the right hypochondriac region, the temperature, and pulse. Wait until the pulse rate is reduced. If rigidity persists to a marked degree, operation should be delayed, but if there is subsidence in physical signs it is perfectly safe to operate. We are absolutely and unalterably opposed to immediate operation in cases of acute cholelithiasis and acute empyema. Properly to handle these cases it is absolutely necessary to hospitalize them at once, where a careful and strict regimen can be instituted. Absolute withdrawal of food and drink are essential so that the intestinal tract is put at rest. Just enough morphine is given to control the pain. Five hundred to 1,000 cubic centimetres of 10 per cent. glucose are given intravenously, 5 per cent. by hypodermoclysis to prevent dehydration and also to maintain the glycogen reserve in the liver. Water by mouth is permitted in twenty-four to forty-eight hours provided the symptoms subside. In addition, daily infusion of glucose is given even after the symptoms have subsided because the reserve stored up by the administration of glucose cannot be overestimated. Calcium has been found of practically no use in the preparation of these cases and therefore has been discontinued. Blood transfusions are substituted and given whenever necessary.

Perforation of the Gall-bladder.—Mentzer believes in immediate surgical intervention. He has shown from statistics taken from the literature that eight cases died while treated medically; that thirty-one perforated while being watched. The danger of perforation of the gall-bladder due to waiting for symptoms to subside is somewhat overestimated. Perforation of the gall-bladder in my experience is a rather rare complication. It cannot occur if the case is under absolute control of the surgeon and this can really be obtained only if the case is hospitalized and the treatment as outlined in the previous paragraph is strictly adhered to. The surgeon must be absolutely firm in insisting upon abstinence from any food, including water. If, however, the patient chooses to stay at home, surrounded by sympathetic family and friends, food and drink will be given with the consequent danger of complications. Physical and physiological rest are essential as a preventive to a perforated gall-bladder. In over twenty-five years of active surgical experience I have observed one perforation in a newly formed gall-bladder following the operation of cholecystectomy. In this case the gall-bladder was as large as the original gall-bladder except that the characteristics of the normal mucosa were absent. The patient recovered.

Anæsthesia.—Spinal anæsthesia has been a great help in reducing morbidity and mortality in operations on the gall-bladder. There is no one factor that has contributed more to the ultimate recovery of the patient than that of spinal anæsthesia. Since using it I have not experienced any of the so-called chemical deaths following cholecystectomy which I believe are in a measure

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due to the rough handling of the liver occasioned by rigid abdominal walls, and possibly the toxic effects of the anæsthetic on the liver. The relaxation induced by spinal anæsthesia allows one to manipulate the field of operation carefully, the dissection of the ducts and blood-vessels is facilitated more readily if one performs the so-called "open operation." By this I mean the visualization of the field in the region of the foramen of Winslow by opening the right free border of the gastrohepatic omentum. One must be careful after this is done that the cystic duct is separated from the common duct because with the relaxation due to spinal anæsthesia, ligation and destruction of the common duct can easily follow. There is nothing in the whole realm of surgery more embarrassing than the ligation and excision of this structure.

Drainage.—Drainage of the field of operation after cholecystectomy is unnecessary in 98 per cent. of the cases. Our guide for not using drainage is a field clear of blood, oozing and the escape of bile. The convalescence is easier, there is less disturbance due to nausea, and gas symptoms are few. The danger of hernia is practically eliminated on account of the omission of drainage and the use of spinal anæsthesia. Not one case undrained had to be reopened for a secondary collection; in fact, the only cases that required re-operation for a subhepatic or subphrenic abscess were those cases that were drained. In my monograph on the subject of gall-bladder I advocated drainage in every case. For several years now I have practically stopped its use, with better results.

Secondary Operations.—Secondary operations should be avoided whenever possible. Cases of acute empyema of the gall-bladder usually require two operations because at the first operation the gall-bladder is drained, leaving a diseased structure that may cause considerable trouble. Secondary operations a few weeks after the primary operation are more dangerous on account of the adhesions which form quickly in all upper abdominal work. If we allow the symptoms to subside a primary cholecystectomy can be performed without danger.

Review of Literature.—H. B. Stone and J. C. Owings,¹ it seems, have the best results from the immediate operation. They are dogmatic concerning the best way to handle the acute cases. They mention many authorities for and against immediate operation. Their mortality is not mentioned but they quote Mentzer who reported 33 per cent. died as a result of late operation, while 19 per cent. died as a result of early operation. Zininger is not able to judge which recover or which grow worse. He states 6.6 per cent. died after waiting five days for delayed operation and 25 per cent. died after immediate operation.

Morris K. Smith² is against immediate operation because of an unfortunate death occurring forty-eight hours after a partial cholecystectomy. This again confirms my observation, that a complete operation cannot always be performed in acute cases on account of the intense inflammation encountered especially at the cystic duct. A second operation for overlooked stones lurking in the piece of remaining gall-bladder, or for a persistent fistula, thus

becomes imperative. Smith quotes statistics from mortality records of cases of acute gall-bladders operated upon. Miller had 13.5 per cent., Whipple 13.7 per cent., H. F. Graham 6 per cent., and Zinninger 7.8 per cent. mortality. Out of 1,053 cases operated on, the mortality in acute cases was 9.3 per cent. and the subsided ones 5.3 per cent.

While Judd and Phillips³ believe in early operations, they admit that not every case should be operated on at once. This I believe will be subscribed to by all surgeons except a radical few. Each case must be treated individually on its own merits. According to Love the mortality in operating on acute gall-bladder is 21 per cent. Love states that one of the dangers of allowing the case to subside is that the patient may leave without operation. This is true, yet it has also been my experience that symptoms will return sooner or later, when the patient is only too willing to undergo operation. The greater danger, to my mind, is the mistake in diagnosis that may occur, confusing the acute gall-bladder with a high appendix, perforated ulcer, and acute pancreatitis. This is plausible but with careful observation I believe the alert surgeon will soon apply the proper remedy, whether that be a waiting policy or immediate operation. Only recently a patient presented himself in the Philadelphia General Hospital with upper abdominal symptoms after being sick at home two days. We observed him for a few hours. The symptoms did not subside, operation was advised; acute pancreatitis was found. In a discussion following the aforesaid papers, Finney summed up the situation cryptically when he said that "one cannot be absolutely dogmatic in any surgical question. . . . One should always exercise surgical judgment and be guided by it."

Harvey Smith,⁵ in a paper read before the Medical Society of the State of Pennsylvania, reported a mortality of 10 per cent., eight out of forty-eight cases of acute gall-bladders operated upon.

Many more references could be given to show that the mortality is greater after operation on acute conditions of the gall-bladder but there can be no better indictment against the immediate operation than the quoted statistics.

Citation of Cases.—A recital of a few cases will help to substantiate the points I wish to make. About four years ago a prominent surgeon from Pennsylvania consulted me for symptoms referable to the upper right abdomen. A diagnosis of empyema of the gall-bladder was made. He wanted me to operate on him at once, which I refused to do. His persistence, however, prevailed upon my better judgment, the operation being performed before the subsidence of symptoms. In this case drainage was used. He lived four days following operation, death being due to a profound toxæmia.

A young woman suffering from acute cholecystitis, with a stone in the cystic duct, was seen in consultation. Curiously enough these cases usually have a single choledochal stone blocking the duct. Since I was leaving for my vacation the following day, the family physician implored me to operate immediately. This was done next morning. She lived twenty-four hours following a high temperature, resembling the fatal cases due to a chemical intoxication.

An interesting case was operated on recently in two stages, three months intervening between operations. The patient never had any symptoms of gall-bladder disease until he suffered an attack of influenza four weeks prior to the acute gall-bladder symp-

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toms. He was unconscious when taken to the operating room. He was operated on in bed under local anaesthesia because of his huge physique, and the fear that too much handling of the patient would be detrimental to his recovery. A diagnosis of a subphrenic collection, and empyema of the gall-bladder was made. A lateral incision was made, a rib resected and the subphrenic space was drained. Believing that this was not the sole source of trouble, the incision was continued over the abdomen and the acutely inflamed gall-bladder exposed to the "side-door" route. The gall-bladder was drained and much pus was obtained. Quite against our expectations the patient recovered; he was able to leave the hospital, and returned for a cholecystectomy which was performed through the original incision three months after the first operation. This case illustrates how an acutely inflamed gall-bladder can cause a profound toxæmia. The patient made a fine recovery.

A woman was admitted with symptoms of acute gall-bladder disease; in addition she had some myocardial degeneration, a not-infrequent accompaniment of biliary tract disease. She was not operated on until a month after admission to the hospital. The gall-bladder was then removed with ease. She recovered from operation, although her pulse was never under 100.

A perplexing problem often presents itself in the pregnant woman. The same waiting policy is followed. One case was pregnant six months with symptoms of empyema of the gall-bladder. After subsidence of symptoms she was operated on and made a good recovery. She did not abort. Another woman one week before delivery had severe symptoms of acute cholecystitis. We waited for two weeks after labor before removing the gall-bladder. Her convalescence was uneventful.

Primary Plans.—Finally, there is no question in my mind that, as a result of immediate operations on acute conditions of the gall-bladder, the mortality will be higher.

The waiting period must vary because some patients respond to treatment more readily than others. Depending on the case, operation can be performed in two days to two weeks or more with greater safety.

Absolute abstinence from food or drink must be strictly enforced. I have yet to see an acutely inflamed gall-bladder fail to respond to this treatment. Glucose by all methods must be used, before and after operation. The glycogen reserve must always be maintained. Perforation need never be feared if this treatment is followed. That we have not had a death from cholecystectomy in over twenty-seven months is largely due to the use of spinal anaesthesia. Statistics prove that the mortality is greater after early, immediate, or emergency operations for acute cholecystitis.

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PRIMARY CARCINOMA OF THE COMMON BILE-DUCT*

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PRIMARY carcinoma of the common bile-duct presents a difficult clinical problem. As new growths in this location early interfere with the function of the duct by partially or completely occluding its lumen, giving rise to definite clinical manifestations while the growth is yet localized, one might suppose surgical intervention would be especially favorable for this type of neoplasm. There are, however, certain inherent difficulties in the diagnosis and treatment of these cases which tend to make the prognosis unfavorable and a study of the reported cases shows that the surgical results in this type of malignancy have not been encouraging.

Renshaw¹ reports a series of twenty cases from The Mayo Clinic covering a period of seventeen years with an operative mortality of 33.3 per cent. The average post-operative length of life of thirteen patients of the series was a little more than five months. Of eighteen operations, four were radical, three were exploratory and eleven palliative.

In Wahl's² six cases of carcinoma of the biliary tract, four were in the extrahepatic biliary ducts exclusive of the gall-bladder and all died shortly after operation, of complications the result of biliary obstruction.

Springer³ mentions a case of carcinoma of the papilla of Vater in the service of Dr. A. V. Moschowitz at the Mount Sinai Hospital in which the tumor was removed by transduodenal circular excision. The patient died on the sixth post-operative day of hæmorrhage. However, he cites another case reported by Dr. DeWitt Stettin in which the patient was alive eight and one-half years after an operation of this type.

Cheney⁴ described a case of primary carcinoma of the common duct confined within the lumen of the duct which was operated upon and a cholecystoduodenostomy performed. The patient died a few days following the operation. The cause of death was not given.

Cabot⁵ reports the case of a male, aged seventy-five years, with jaundice of only a few days' duration. A pre-operative diagnosis of carcinoma at the head of the pancreas was made. At operation, a rounded mass was revealed beginning in the hepatic duct just proximal to the junction of the cystic and common ducts and extending into the common duct. A cholecystogastrostomy was done, which, of course, did not relieve the obstruction. The patient died about two weeks later. Bronchopneumonia and severe hæmorrhage were post-operative complications.

In the group of cases collected by Quenu in which radical operation was performed, there were twelve post-operative deaths.

Although primary carcinoma of the common bile-duct is a comparatively rare pathological entity, it should be given consideration as a possible cause of persistent obstructive jaundice in all cases occurring in patients past middle age.

It is with the view of emphasizing the particular difficulties involved in

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the diagnosis and treatment of common-duct obstruction due to primary carcinoma that the following two cases are reported.

CASE I.—(No. 83467) E. M., a white female, aged fifty-four, admitted to the Graduate Hospital March 15, 1930, with the history that three weeks before admission she developed generalized itching. In a few days she became jaundiced. The stools were clay colored and the urine dark. These symptoms persisted and were present on admission. She had had no pain at any time. She was an obese female who did not appear acutely ill. Skin and sclerae had a deep icteric hue and the body showed scattered purpuric spots. Edge of liver palpable 12 cm. below the costal margin, smooth, rounded, no nodules felt. The spleen was palpable. No masses or areas of tenderness. X-ray studies of the gastro-intestinal tract revealed rapid emptying time of the stomach and duodenum, suggesting extrinsic irritation.

Laboratory Studies.—Wassermann and Kahn negative; icterus index 120; van den Bergh direct immediate, indirect varied from 2.8 to 10.5; blood sugar 80 mg. per 100 cc. of blood; cholesterol 290 mg.; urea nitrogen 13 mg.; calcium 9 mg.; urobilinogen 1-20 to 1-50. Coagulation time 5.5 min.; bleeding time 2.5 min. Bromosulphthalein 2 mg., 30 min. 40 per cent., 60 min. 25 per cent. Erythrocytes 4,130,000; polymorphonuclears 63 per cent.; small lymphocytes 37 per cent.; platelets 408,000. Fragility test, hæmolysis began at 0.40 per cent.; complete at 0.28 per cent. Urine negative. Stools negative for occult blood. Temperature and pulse range were normal.

The diagnostic possibilities were five: (1) catarrhal jaundice; (2) stone in common duct; (3) carcinoma of head of pancreas; (4) cancer, primary elsewhere, with secondary involvement of the liver and bile-ducts; (5) the clinical course of rapid, silent, persistent jaundice with a palpable liver edge that is not nodular, but smooth and rounded, was compatible with, and suggestive of, carcinoma of the common bile-duct.

In view of these considerations, the patient was referred to the surgical service for operation to relieve the existing obstruction, which was performed April 4, 1930, under spinal anaesthesia. Upper right rectus incision. Gall-bladder was found shrunken and collapsed, and contained very little bile and no stones. A thickened fibrous area was felt in the common duct which extended from above the junction of the cystic and common ducts to well below this point in the common duct. The duct was opened by a longitudinal incision and a definitely strictured area was exposed which gave one the impression of scar tissue, the result of previous trauma or ulceration. There was no suggestion of tumor formation and the regional lymph-nodes were not visibly enlarged. Exploration of the entire duct with a probe toward both the duodenal and hepatic ends failed to demonstrate stones or other pathology. Assuming that the stricture was benign, a restoration of the lumen of the hepatic and common ducts was attempted with a No. 12 F. soft rubber catheter. Two cigarette drains were introduced into Morrison's pouch and the abdominal wall was closed in layers about these drains.

Convalescence was smooth until the fifth post-operative day, when bleeding began. The dressings were saturated with blood and considerable blood was passed in the form of tarry stools. The coagulation time and bleeding time were within normal limits. Two hundred and fifty cc. of a 5 per cent. glucose solution were given intravenously every twelve hours and 10 cc. of 5 per cent. calcium chloride. Two transfusions of citrated blood were given. Cardiac failure threatened from the loss of blood volume and in addition to replenishing the fluid volume, large doses of tincture of digitalis were given. On the seventh post-operative day the hæmorrhage recurred to an alarming degree. Bleeding occurred freely from the abdominal wound and much blood was passed in the stools. The patient went into a state of shock. She was taken to the operating room and two deep mattress sutures were passed through the abdominal wall to control the hæmorrhage. A transfusion of 500 cc. of citrated blood was given and 200 cc. of normal saline. These measures apparently controlled the visible hæmorrhage and the patient recovered from the shock. However, bleeding persisted from the gastro-intestinal

tract. She was again transfused with 500 cc. of citrated blood and 200 cc. of normal saline. She failed to rally, however, and died shortly following the last transfusion.

Pathological Notes.—The autopsy was limited to the abdomen. There was a considerable amount of bile-stained blood in the abdominal cavity. Outside of the fact that all the tissues were bile-stained, the pathology was confined to the bile-duct, liver and pancreas. Upon opening the common bile-duct, the catheter was seen to be in place. The fibrous stricture in the duct extended from 1.5 cm. above the junction of the cystic and common ducts to well below this level. The wall of the stricture was 7 mm. thick and 2 cm. long. Two fistulae were seen 1 cm. proximal to the origin of the cystic duct, through which bile had escaped into the peritoneal cavity. The leakage had occurred along the suture line. The superior surface of the liver was adherent to the diaphragm by numerous adhesions, easily broken. Pressure exerted upon the gall-bladder permitted bile to flow into the duodenum.

Microscopical Diagnosis.—Bile-duct: Adeno-carcinoma and recent thrombosis. Liver: parenchymatous degeneration of cells, chronic passive congestion, perihepatitis. Pancreas: chronic pancreatitis, hyperplasia of stroma.

CASE II.—(No. 83898) A. R. A white female, aged forty-eight, admitted to the Graduate Hospital April 3, 1930, in the metabolic service of the late Dr. O. H. Petty. She had had diabetes for the previous six months, but had not consistently adhered to diet or insulin treatment. Seven weeks before admission she had an attack of nausea and vomiting accompanied by pain in the gall-bladder region which was referred to the right shoulder and back. The pain lasted for two days. She became jaundiced. The jaundice gradually disappeared. About three weeks later she had a similar attack which required a hypodermic of morphine for the relief of the pain. The jaundice had persisted from that time, with recurring attacks of nausea and vomiting. No recurrence of pain. She had grown weaker and was losing weight rapidly. On admission she was deeply jaundiced and greatly emaciated. There was tenderness in the region of the gall-bladder. No other organs or masses were palpable.

Early operation was advised in order to relieve the obstructive jaundice as soon as possible. The obstruction was considered particularly harmful in view of the diabetes.

April 4, 1930, under spinal anaesthesia, the abdomen was opened through an upper right rectus incision. All the organs and tissues of the abdominal cavity were deeply jaundiced. The liver was greatly swollen, congested, the edges rounded and there was evidence of acute hepatitis. The gall-bladder was enormously distended, its walls thickened and opaque. The common and cystic ducts were carefully palpated, but no definite stone was felt. The common duct was not distended. The distended gall-bladder was opened and a perfectly clear liquid escaped. A finger was then placed in the foremen of Winslow and the common and cystic ducts milked upward in an effort to palpate stones. Immediately a purulent bile-stained fluid escaped from the opening in the fundus of the gall-bladder and with it a soft, muddy, black, gall-stone debris. Most of the gall-stone debris was small, about the size of sand granules, although some of the pieces were as large as a French pea. This milking process was repeated several times until no more gall-stone debris could be obtained. A spoon curette removed a number of pieces of gall-stones larger than previously found. A probe was passed through the dilated cystic duct, into the common duct, toward both hepatic and duodenal ends without encountering resistance. Although the common duct had been thoroughly palpated, no evidence of malignancy was discovered.

The condition of the patient was such that any extended operation was considered inadvisable. Drainage of the gall-bladder was decided upon with the hope of re-operating when the patient's condition had improved. The exploration had been as thorough as could be done without opening the common duct or duodenum. The post-operative course was very stormy. Vomiting was frequent and difficult to control. It was practically impossible to keep a duodenal tube in place. Large amounts of fluids and glucose were administered by hypodermoclysis and intravenously to combat dehydration. During

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the entire period the drainage through the cholecystostomy tube was free, so free in fact, that it seemed an obstruction in the common duct still existed. Bile drainage varied from 500 cc. to 2,500 cc. per twenty-four hours. During the period when there was an excessive loss of bile an attempt was made to return it by way of the duodenal tube. This was only partly successful because of the difficulty of retaining this tube in the duodenum. The bile then was given by proctoclysis. The patient remained markedly jaundiced throughout her illness.

In spite of all supportive measures the patient's condition rapidly declined and she died April 25, 1930, the twenty-first post-operative day. The autopsy was confined to the abdomen. Stomach: Distended with fluid and gas. Duodenum: The valvulae conniventi prominent. This structure contained mucus. Around the papilla of Vater there was an ulcerated area with elevated edges. This surrounded the opening of the common duct and the duct of Wirsung. Obstruction had taken place, for both of these ducts were very much dilated. The common bile-duct was fully 1.5 cm. in circumference and the pancreatic duct twice its normal size. *Diagnosis:* Carcinoma involving the submucosa near the ampulla of Vater; chronic interstitial pancreatitis; cirrhosis of liver, hepatitis. Gall-bladder: Small and contracted. There was an opening in the fundus through which it drained. The cystic duct was not dilated to any great extent, while the hepatic duct showed dilation.

The history of rapid onset of a progressive and finally complete jaundice with the physical findings in these two cases pointed rather strongly toward a malignancy although in the second case the history of two acute attacks of pain suggested the presence of gall-stones. However, it was considered possible for a neoplasm of the common duct to stimulate a stone by causing spasm of the duct with resultant acute attacks of biliary colic. The pre-operative diagnosis of malignancy causing obstruction made in both instances was changed at the operation to benign stricture, and cholelithiasis respectively, only to have the pre-operative diagnosis confirmed at autopsy. It is only fair to say, however, that in Case II, a more thorough investigation was impossible because of the patient's critical condition.

These cases emphasize in no uncertain manner the ease with which a small growth confined to the common duct may be mistaken or missed at operation. It leads one to suspect that many cases of primary malignancy of the common duct may be missed entirely unless the surgeon is particularly alert to detect them and makes a thorough exploration of the common duct by choledochotomy or transduodenal exploration of the papilla and ampulla in every suspicious case. Such cases may go entirely unrecorded unless they come to re-operation or autopsy.

It is important to realize, while exploring the common duct, that a strictured and fibrous area may harbor malignant cells without having the gross appearance of a tumor. This fact was well emphasized in our first case.

The cause of death in Case I was entirely due to post-operative hæmorrhage, incident to the biliary obstruction. It could as well have been due to biliary obstruction from any cause. The rapid and fatal termination after the onset of the hæmorrhagic state demonstrates conclusively that there is a certain type of post-operative hæmorrhagic diathesis following obstructive jaundice for which there is as yet no adequate treatment.

The coagulation time and bleeding time proved not only worthless but misleading as as indices of the hæmorrhagic tendency. It is hoped that the sedimentation rate, as suggested by Linton,⁹ may prove of prognostic value in these cases.

The cause of death in the second case was grave metabolic disturbance, the result of diabetes, plus renal and hepatic insufficiency. The loss of large amounts of bile may have been a contributing factor to the metabolic upset, but this was in part compensated for by the re-administration of bile by tube. The biliary obstruction together with the renal and hepatic insufficiency made the diabetes especially malignant.

Etiology.—That gall-stones and chronic cholecystitis constitute a pre-cancerous condition of the gall-bladder is generally held. Miller,⁷ in a study of operative and autopsy records of over 10,000 cases of gall-stones, found an incidence of 4.1 per cent. of carcinomas. The relationship of gall-stones to cancer of the bile-ducts, however, does not appear to be direct. Gall-stones are more prevalent in the female, while cancer of the bile-duct is almost equally distributed with possibly a slight preponderance in the male. Infection or chronic irritation may be an etiological factor.

Symptoms.—The possibility of primary carcinoma of the common bile-duct should be considered in all cases of obstructive jaundice which persist for over a period of two or three weeks in a patient at or past middle age.

The occurrence of biliary colic in obstructive jaundice does not necessarily rule out the chance of a primary malignancy being present. Gall-stones causing partial or complete obstruction may co-exist with a primary new growth. Also a new growth, by setting up an irritative spasm of the bile-duct, may cause acute biliary colic. There are several recorded cases of co-existing gall-stones and primary malignancy in which the clinical and operative findings led to a diagnosis of cholelithiasis, with the later finding of a tumor at re-operation, or autopsy. In the absence of biliary colic, a history such as the following is very suggestive; the development of a rapid, complete non-febrile jaundice in a person of about middle age, which has lasted long enough to exclude catarrhal jaundice, and associated with a certain amount of epigastric distress. Confirmatory physical findings will be the smooth rounded edge of the liver palpated a few centimetres below the costal margin and a palpable distended gall-bladder which is not tender.

Diagnosis.—The diagnosis of biliary obstruction is not difficult. This is the important finding. An afebrile jaundice with high readings of the icterus index and van den Bergh, dark urine, clay-colored stools, an absence of urobilin in the urine and faeces, and an absence of bile in the duodenum makes the condition self-evident.

Early recognition as a surgical condition and early operation, with complete exploration, is the only means of making certain the presence or absence of malignancy in early cases. Stones, as a rule, do not completely obstruct the common duct. Urobilin in the urine and faeces indicates that some bile passes into the duodenum. Analysis of duodenal contents will determine whether obstruction is complete or not. Tumors, on the other hand, are more apt to cause a complete obstruction. Rarely, jaundice may be absent during the entire time, as in the cases reported by Cabot⁵ and Walters.¹⁶ The possible co-existence of gall-stones and tumors should always be remembered. Courvoisier's law is not of any particular aid in making a diagnosis of the individual cases.

Prognosis.—The prognosis, generally speaking, except for the complications resulting from biliary obstruction, should be better than for malignancies located elsewhere in the gastro-intestinal tract and associated organs because a growth here declares itself early, by mechanical blocking of the duct. When

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the growth is confined to the duct, the prognosis, in a large measure, is dependent on the complications resulting from biliary obstruction and naturally the longer the jaundice has been present the poorer the prognosis will be.

Pathology.—The usual site of cancer of the common duct is either at the junction of the hepatic and cystic ducts, or near the duodenal termination of the common duct, where stagnation of bile or trauma are most apt to occur.

Rolleston,⁸ in a study of 100 collected cases, found that one-third of all new growths of the bile-ducts arise in the lower terminal portion of the choledochus. The origin of new growths in this region may be from: (1) common bile-duct; (2) ampulla of Vater; (3) duct of Wirsung; (4) papilla of Vater; (5) duodenal mucosa; (6) head of the pancreas. The most common type is a growth arising from the duodenal surface of the papilla of Vater. It usually takes the form of an eroded ulcer with a depressed centre, and over-hanging thickened carcinomatous walls. The distribution of Rolleston's⁹ 100 cases was: Common duct, forty-four; junction of the three ducts, twenty-seven; hepatic duct, twenty-two; and cystic duct, seven.

They occur usually in three forms, namely, (1) villous growths which may distend and fill the ducts; (2) nodular masses which tend to encroach upon and constrict the duct; (3) a diffuse growth along the duct which converts it into a firm tube. The usual form is adeno-carcinoma.

Rolleston⁹ believes the majority of carcinomas of the bile-ducts are derived from the surface epithelium although the spheroidal cell carcinoma may be derived from the mucous glands in its wall. An analysis of forty-three cases showed thirty-seven columnar; five spheroidal, and one colloidal in type.

Mucoid degeneration of the columnar cells, or transition from columnar to spheroidal cells may take place, and metaplasia may result in the formation of squamous-cell carcinomata. According to Ziegler,¹⁰ MacCallum¹¹ and Ewing,¹² carcinoma of the bile-ducts is usually of the columnar-cell type. Mayo Robson¹³ states that carcinoma at the head of the pancreas is spheroidal cell, while carcinoma of the ducts is almost always columnar in type.

The one striking gross pathological feature is that they are often seen in the early stage, because of the mechanical interference which they create, due to their strategic position, while they are yet so small as to be easily overlooked at operation or autopsy.

Benign tumors of the extrahepatic ducts are comparatively rare; Bazin¹⁴ reported a case of papilloma of the common duct in which successful excision of the tumor was done. He cites sixteen cases gathered from the literature, some of which are on the border-line of malignancy. He states that all papillomata are potentially malignant.

In a series of tumors of the bile-ducts, reported by Marshall,¹⁵ there were four benign, as compared to forty-nine malignant. These cases were gathered from operative material at The Mayo Clinic, extending over a period of twenty years, and comprising 23,000 operations on the biliary apparatus.

From this wealth of material the following observations are made:

- (1) Prevalence of carcinoma of the bile-ducts in patients past middle age.
- (2) Prevalence in the male, 62 per cent.
- (3) Relative low-grade malignancy of the tumor with considerable amount of fibrous tissue.
- (4) The predominant tumor is adeno-carcinoma composed of columnar epithelium.

(5) Hæmorrhage is the most common serious post-operative complication and the most common cause of death.

From the practical standpoint the distinction between benign and malignant tumors of the common duct is more of academic than clinical interest, as on the one hand, the malignant growths are usually small, confined to the duct, slow-growing, and slow to metastasize, and on the other, the benign tumors are potentially malignant, and are often border-line.

In both types mechanical interference is responsible for the disturbed chemical and physiological processes, expressing themselves clinically in hæmorrhage and cholæmia.

Treatment.—The pre-operative preparation consists of:

(1) Administration of large amounts of easily assimilated carbohydrates to build up the glycogen reserve of the liver.

(2) Adequate fluid intake of at least 3,000 cc. of fluid daily. This may be administered in the form of a salt solution by hypodermoclysis or infusion, if there is a tendency towards vomiting.

(3) Calcium in some form, either as calcium chloride by vein, or as calcium lactate by mouth, should be given daily for a few days pre-operatively, in the effort to prevent the post-operative hæmorrhagic tendency, although recent studies would make it appear that a lack of available calcium is not a factor in the hæmorrhagic tendency in obstruction jaundice.

(4) Transfusion of whole blood is indicated as a preparative measure when the hæmorrhagic tendency is manifested by purpuric spots or a rapid sedimentation rate is present.

Operative Procedure.—Spinal anæsthesia seems to be particularly well suited to this type of case, as it spares the liver the burden of a general anæsthetic and makes easy the exposure of the hepatic and common ducts with a minimum of trauma. Palliative operations of cholecystostomy or cholecystenterostomy may be indicated when the lesion is below the level of the cystic duct, and is irremovable or where a more extensive and radical procedure is contemplated at a second operation after improvement of the patient's condition has taken place.

Radical operation varies with the type of pathology existing. A three-stage operation has been suggested by Kansch¹⁷ for tumors at the lower end of the common duct. The first stage consists of a preliminary cholecystenterostomy to relieve the jaundice and improve the nutrition. At the second stage, radical excision of the tumor is done. Adjacent portions of the duodenum and head of the pancreas are resected if necessary with blind closure of the upper end of the duodenum, implantation of the ablated head of the pancreas, common bile and pancreatic ducts, into the lower end of the duodenum. A gastroenterostomy is done at the third stage.

Where the growth is strictly confined to the papilla, simple excision may be done through a transduodenal incision. With the growth located higher

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in the duct, but below the cystic duct, excision of that portion with cholecystogastrostomy or cholecystenterostomy may be done.

When the growth is at the level of the cystic duct or higher, resection of the involved portion with implantation of the hepatic duct into the stomach or duodenum is the procedure of choice, but considerable technical difficulty may be encountered.

In the series reported by Renshaw,¹ resection of a portion of the duct with end-to-end union was done in one case, and resection of a portion of the wall with a plastic repair of the duct over a T-tube was done in another instance.

Post-operatively prompt measures are indicated to support the liver metabolism by the use of glucose and fluids. Digitalis may be indicated for the circulation. Blood transfusions should be resorted to where a hæmorrhagic tendency is present. Calcium chloride and parathormone may be of some use. Re-introduction of bile by duodenal or rectal tube is indicated where bile loss is excessive. The continuous intravenous drip of 5 per cent. glucose in Ringer's solution as advocated by Matas for various post-operative conditions should prove especially valuable in cases of obstructive jaundice complicated by hæmorrhage and hepatic insufficiency.

CONCLUSIONS.—(1) The symptoms of primary cancer of the common bile-duct are not pathognomonic. They may be suggestive but in final analysis they are simply the symptoms of mechanical biliary obstruction.

(2) It is important to consider the possibility of a primary malignant growth in all cases of persisting obstructive jaundice in patients past middle life. The presence of one or more gall-stones in the common duct should not lead one to neglect a complete exploration of the ducts.

(3) Early operation is urged with thorough and painstaking exploration of the common duct for possible malignancy in all suspicious cases.

(4) The high mortality which attends operations for biliary obstruction due to primary cancer is due almost entirely to complications resulting from mechanical obstruction of the biliary tract.

(5) Post-operative hæmorrhage following the relief of mechanical biliary obstruction is responsible for the high percentage of deaths.

(6) Diabetes complicating complete biliary obstruction is particularly dangerous, due to grave metabolic disturbance.

(7) Early diagnosis and early operation will tend to lower the mortality rate not only by minimizing the chance of extension of the primary growth, but by decreasing the incidence of complications due to obstructive jaundice, namely, hæmorrhage, hepatic insufficiency and cholæmia.

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THE RÖNTGENOLOGICAL LOCALIZATION OF SPINAL SUBARACHNOID BLOCK BY THE USE OF AIR IN THE SUBARACHNOID SPACE

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THE difficulty of determining, at times, the exact level of a subarachnoid block due to tumor and other lesions is too well known to warrant comment. The use of lipiodol or similar preparations has added greatly to the certainty of diagnoses both as to the site of such a block and, to a certain extent, its nature. Where the subarachnoid block is complete or nearly so, the röntgenological demonstration of a few cubic centimetres of air injected in the lumbar subarachnoid space will serve to denote the level of the lesion as well as lipiodol. The meningeal reaction following the injection of a few cubic centimetres of air is practically nil, and certainly far less than that following a lipiodol injection. It also has the advantage of being rapidly absorbed rather than acting as a foreign body for a period of months or longer.

A résumé of three typical case records is given where the exact level of the lesion seemed to be in doubt.

CASE REPORTS.—CASE I.—R. H. (Hosp. Case No. 18,306), male, age thirty-one, was admitted to the Strong Memorial Hospital because of "pain in the left kidney region."

Fifteen months prior to entry the patient had contracted an acute Neisserian infection of the urethra. This was followed by a severe prostatitis and cystitis. Some twelve months prior to entry he experienced urinary retention and from that time on catheterized himself at irregular intervals. A severe pyelitis evidently ensued with considerable backache, pain in the region of the left kidney, chills and fever. Because of this he was confined to another hospital for four months. At about the same time that urinary retention was noted a sense of burning, numbness, and pain of both lower extremities began. Weakness of both lower extremities was also noted. These symptoms progressed until he was barely able to walk or turn over in bed. At the time of entry he was emaciated and practically bedridden. The urinary tract was found to be badly infected. Bladder calculi added to the difficulty of treatment of the infection.

Neurological examination showed an indefinite sensory level at about the second lumbar segment. Knee and ankle jerks were absent. The cremaster reflexes were present. The plantar and anal reflexes were absent. A lumbar puncture showed a slightly xanthochromic fluid. The initial pressure was 52 millimetres of water. There was no rise in pressure on repeated jugular compression. Cell count was five, Pandy plus, Ross-Jones plus. A few well-crenated red cells could also be made out in the spinal fluid. Chloride determination was 695 milligrams per 100 cubic centimetres; sugar, 64 milligrams, non-protein nitrogen 18.7 milligrams. Six cubic centimetres of air were injected into the subarachnoid space after the fluid had been allowed to escape. X-rays taken with the patient in the sitting position showed the air shadow stopping at the level of the body of the second lumbar vertebra. (Fig. 1.)

After the urinary tract seemed to be as free of infection as it might become for some time, a laminectomy was performed. A large tumor ventral to the cauda equina was

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found and removed. Pathologically it proved to be a tumor arising from the perineural sheath of one of the filaments of the cauda equina. (Fig. 2.) The patient did well until the ninth post-operative day when he developed fever and other signs of meningitis. Death occurred three days later. Autopsy revealed a bilateral pyonephrosis, multiple renal abscesses, multiple vesical calculi with cystitis, and a generalized meningitis.

CASE II.—H. F. (Hosp. Case No. 23,874), age, thirty-nine. Increasing numbness and pain in both lower extremities for two years, occasionally pain "between the shoulder



FIG. 1.—Reproduction of X-ray negative showing air in spinal subarachnoid space denoting lower level of subarachnoid block.

blades," and occasionally a sensation of numbness in the right middle finger were the principal complaints. He could barely walk even with the aid of canes. Both lower extremities were spastic. Deep reflexes were hyperactive. Ankle clonus and Babinski's sign were bilaterally positive. Superficial reflexes were absent except pharyngeal and corneal. The sensory level varied from time to time but was never recorded above that of the fifth thoracic segment.

A lumbar puncture showed the spinal fluid to be xanthochromic. Initial spinal-fluid pressure was 130 millimetres water; abdominal compression caused a rise to 190 millimetres water. Jugular compression resulted in no rise in the manometer. A diagnosis of a spinal-cord tumor was made.

DIAGNOSTIC USE AIR IN SUBARACHNOID SPACE

Because of the history of numbness of the right middle finger, a lesion at a higher level than dorsal five or the sensory level, was suspected. Consequently 6 cubic centimetres of air were injected into the lumbar subarachnoid space. Stereo A.P. X-rays were taken which showed the lower level of the subarachnoid block at the body of the second thoracic vertebra. In this instance the air shadow of the trachea overlay that in the subarachnoid space and could not be delimited except in stereo X-ray plates.

A laminectomy revealed a large extramedullary benign tumor (perineural fibroblas-

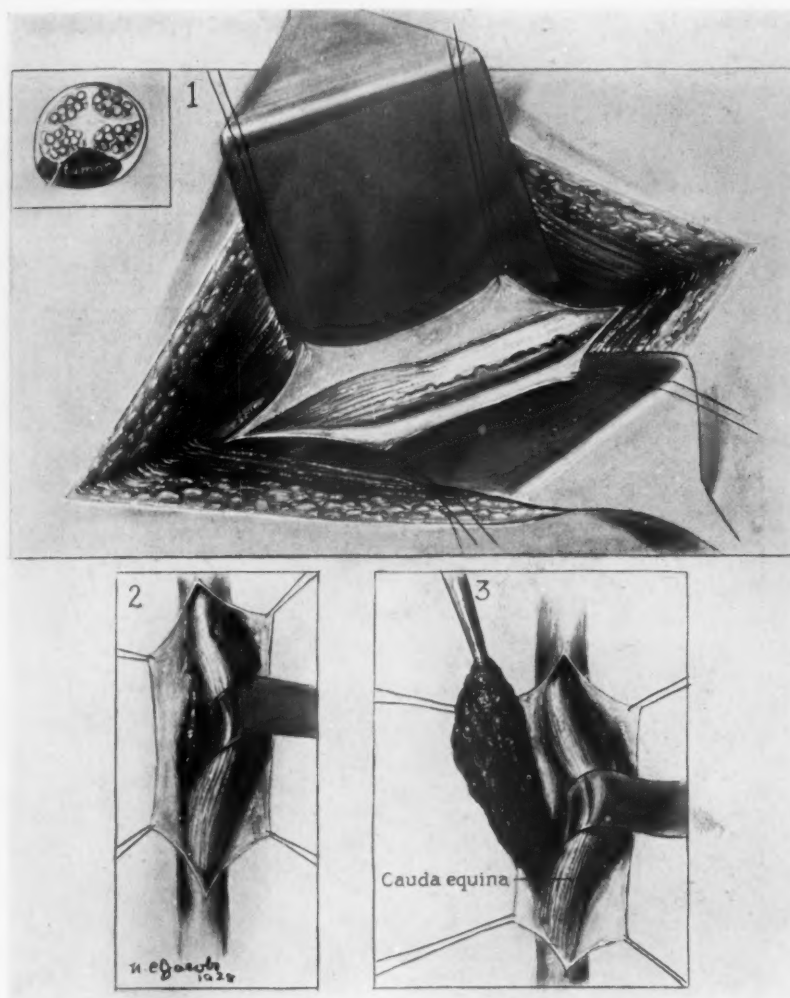


FIG. 2.—Artist's sketch of tumor removed at operation in Case I.

toma) which was removed in toto. (Fig. 3.) The patient has recovered sufficiently so that he can walk easily without canes although signs of permanent pyramidal tract damage persist.

CASE III.—A. S. (Hosp. Case No. 50,026), age forty-three. The exact date of onset of the present illness may have dated back some ten years at which time the patient complained of a dull dragging pain in the left lower quadrant. Two and one half years ago pain in the left lumbar region radiating into the groin was complained of. A left nephrectomy was performed without relief of symptoms. Three months prior to entry



FIG. 3.—Photograph of tumor producing subarachnoid block in Case II.

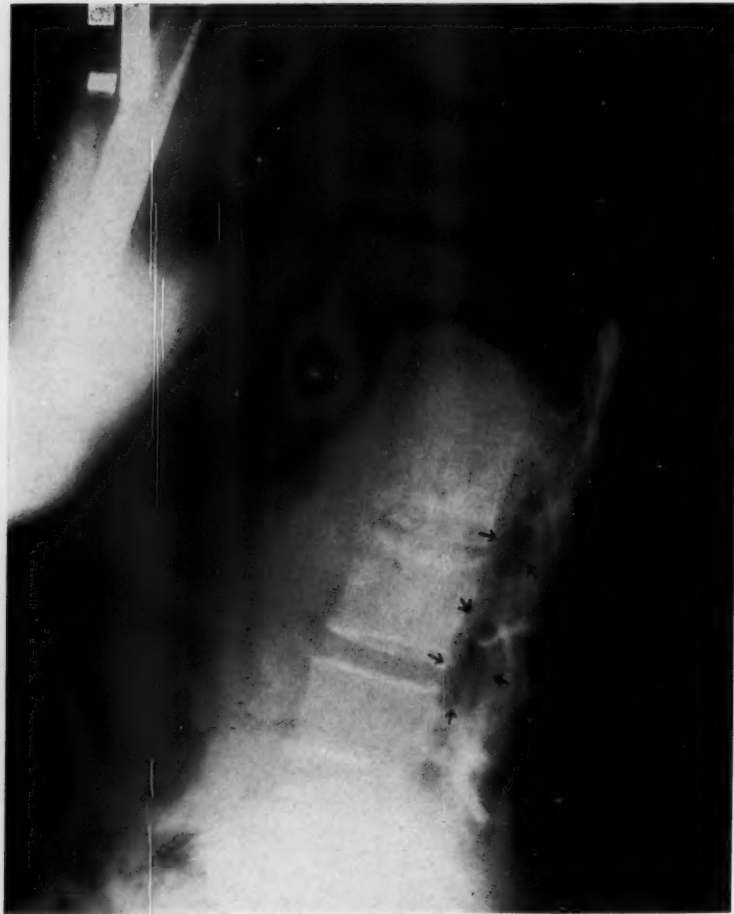


FIG. 4.—Reproduction of X-ray negative showing air in the spinal subarachnoid space denoting the lower level of the subarachnoid block.

DIAGNOSTIC USE AIR IN SUBARACHNOID SPACE

loss of sphincter control was noted. When admitted she presented a nearly complete paraplegia. The sensory level was uncertain and variable. A lumbar puncture showed the spinal fluid to be xanthochromic. Pressure readings gave evidence of a complete spinal-fluid block. Six cubic centimetres of air were injected into the lumbar subarachnoid space. X-ray films taken showed the lower limit of the subarachnoid block to be at the level of thoracic 12. (Fig. 4.)

A laminectomy revealed a large, very vascular extramedullary tumor which had compressed the lower end of the cord into a ribbon-like structure. The intradural portion of the tumor measured some 5 by 6 by 2 centimetres. It was attached to the dura, which was removed with it. The tumor was then seen to be of the collar-button type with an extension ventrally alongside of the body of the eleventh thoracic vertebra. This portion was not removed in as much as it seemed likely that the extent of cord compression was too great to hope for any relief of paraplegia. Histologically the tumor was a hæmangioendothelioma.

Method of taking X-ray films.—All X-rays naturally must be taken with the patient in the sitting position. True lateral films of the spine are preferable to antero-posterior views. In the upper thoracic region stereo A.P. films may be taken if the shoulder-girdle structures obscure the spinal canal.

In two other instances of a complete spinal subarachnoid block air was injected into the lumbar sac to determine the site of the lesion. X-rays failed to demonstrate a collection of air in the spinal canal. Subsequent cisternal lipiodol injections revealed the cause of the block to be a chronic arachnoiditis. This was also verified at operation.

SUMMARY.—The röntgenological demonstrations of air (5 to 8 cubic centimetres) injected into the lumbar space is a simple means of demonstrating the site of a complete subarachnoid block. In instances of chronic arachnoiditis, however, the air is too widely distributed and finely divided to be demonstrated röntgenologically.

The advantage of a lumbar air injection over lipiodol injected into the cervical subarachnoid space is its rapid absorption and the ease and lack of risk of the procedure.

TREATMENT OF TUBERCULOUS EMPYEMA COMPLICATED BY PYOGENIC INFECTION*

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TUBERCULAR infection of the pleura has been universally recognized as one of the causes of chronicity in empyema. It never was an unusual or rare condition. It has become much more common since the introduction and extensive use of artificial pneumothorax in the treatment of pulmonary tuberculosis.

The procedures designed for the treatment of chronic pyogenic infections of the pleura, such as the Schede, Estlander and Delorme operations, are not as a rule followed by successful results. On this account tubercular empyemata have been considered incurable. At the present time, however, with the advances in thoracic surgery, these cases may be successfully treated by new procedures especially designed for their care.

The appearance of tuberculous pus during the use of artificial pneumothorax, although disturbing, is not necessarily alarming, because patients will often tolerate large amounts well without presenting any untoward symptoms. The management of such cases will not be taken up at this time but our attention will be directed only to those cases of tuberculous empyema which are accompanied by pyogenic infections.

Such cases present a complicated problem, as there exist two distinct pathological processes demanding our attention. The proper treatment of the one, pyogenic infection, may be contrary to the most desirable procedure for the care of the other, namely, the tuberculous infection. Often the tubercular nature of the case is not recognized or even suspected until an appreciable time after drainage has been established by means of a rib resection.

A knowledge of the changes, which occur in tuberculosis of the pleura, is essential to a proper understanding of its treatment. The pleura may become affected by tuberculosis in two ways: through a blood-stream infection or through absorption of a pleural exudate containing tubercle bacilli. In a blood-stream infection the bacilli may lodge in any portion of the pleura either internal to or external to its elastic layer. After this lodgment true tubercles develop, and the process may progress through the various classical stages of a tubercular process elsewhere in the body including caseation.

The more frequent manner of involvement, however, is through absorption of bacilli from a pleural exudate. In this instance bacilli come from a focus within the lung. Under these circumstances a tubercular pleuritis develops, characterized by the formation of tubercular granulation tissue without the

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development of true tubercles and limited to that portion of the pleura lying internal to the elastic layer, the tubercular process rarely extending external to this layer. Usually this process is accompanied by a profuse pleural exudate of either clear or bloody serum. Should pyogenic organisms gain access to such an exudate the case will present all the appearances of an acute pyogenic empyema, and its tubercular nature will be completely masked. If, however, this primary serous exudate should be aspirated, the two layers of the pleura may become adherent, the pleural cavity become obliterated, and the process remain dormant for years; or, the case may run the course of a pulmonary tubercular lesion. Likewise after aspiration the exudate may reaccumulate, alter its character and gradually become more and more purulent, until a true tuberculous empyema is established.

Coincident with this alteration in the character of the exudate the parietal pleura undergoes profound pathological changes. The visceral pleura, on the other hand, never alters to the same degree. These changes in the parietal pleura are characterized by the development of tubercles internal to the elastic layer and the formation of non-tubercular fibrous tissue external to the elastic layer. This fibrous tissue may accumulate to a thickness of one or more centimetres. In cases of long duration it is not unusual to have calcareous plaques form throughout the pleura. These are usually limited to the inner surface but rarely may involve the entire thickness of the pleura.

The pyogenic organisms may gain access to the pleural cavity in these cases in several different ways. Theoretically, they may enter via the blood- or lymph-stream, although we have no cases in which we have been convinced that this was their mode of entrance. They may be introduced from without through some fault in technic during aspiration. They may arise from the rupture of a small or large tuberculous focus in the lung. The seriousness of infection by this latter path will vary considerably depending on whether a patent pleuropulmonary fistula is or is not established. The presence of a patent pleuropulmonary fistula may alter materially the management of a case.

The organisms which we have recovered from such cases have been various strains of streptococci, staphylococci, pneumococci, the colon bacillus and numerous forms of anaërobes. Some of the latter have developed a foul odor, others have not.

The management of a tuberculous empyema with an infection of pyogenic organisms will be influenced by the severity of the patient's reaction to this pyogenic infection, and our treatment must be directed primarily toward its cure.

There have been a few cases which present symptoms of such profound sepsis, that an immediate drainage by rib resection must be done. A portion of the tenth rib in the scapular or posterior axillary line should be removed. This is a rib or two lower than is usually chosen in operations for an uncomplicated empyema, the tenth rib being selected so that with the subsequent rise of the diaphragm a tract leading into the pleural cavity will be established between the diaphragm and the chest-wall. Such a tract will heal more readily

than those extending directly through an intercostal space. When the sepsis has been controlled we proceed to obliterate the pleural space in a manner to be described later.

Most cases, although thoroughly septic, have not demanded immediate drainage by rib resection, and we attempt to eliminate the pyogenic organisms by irrigation with antiseptic solutions. We have employed various substances for this purpose, none of which has proved consistently satisfactory. A 1 per cent. watery solution of gentian violet or crystalline violet, a purer form of the same substance, has given the best results. Doctor Amberson and Doctor Riggins have perfected the technic and will at some future date publish in detail their results. At this time it may be stated that the pyogenic organisms have been eliminated in a certain proportion of cases and in other cases the violent nature of the infection has been controlled sufficiently to allow us to proceed with the obliteration of the pleural space. The anaërobic organisms are more resistant to this form of treatment than are the ordinary pyogenic ones, and in the presence of a persistent pleuropulmonary fistula it has not been possible to render the exudate sterile. This is no doubt due to the constant reinfection from the focus within the lung.

The procedure in these cases is to aspirate the pleural exudate and replace it by an equal amount of air. A pyopneumothorax is thus established if one did not previously exist. Ten cc. of a 1 per cent. watery solution of gentian violet is left in the pleural cavity. Following this injection the exudate becomes a thick jelly which must be removed by irrigation with normal saline solution on subsequent days, and fresh gentian violet solution injected and left in the pleural cavity.

If it is not possible to control the septic manifestations promptly, a catheter is introduced through the tenth interspace by means of a trochar and cannula, and the accumulation of exudate prevented through drainage.

After the pyogenic infection has been controlled, an extrapleural thoracoplasty is done in order to obliterate the pleural cavity. The state of nutrition of the patient and any accompanying lesions which might serve as contraindications must be considered. Experience and judgment alone can determine the optimum time for operation.

The operation of extrapleural thoracoplasty is not a technically difficult one, and every well-trained surgeon should be able to perform it.

There are, however, certain features which are important and must be carried out to insure success. The procedure must be performed in several stages, and a few ribs only should be removed at any one time.

We employ a hockey stick or right-angled incision, the vertical limb extending 2 cc. from the vertebral spines and the horizontal limb coming off at almost right angle extending forward to the mid-axillary line. We always remove the uppermost ribs at the first stage. The ribs are removed at their articulations with the tip of the transverse processes. In those cases in which the lung is completely collapsed against the mediastinum the transverse processes are removed and also the rib as far posteriorly as its neck.

TUBERCULOUS EMPYEMA COMPLICATED BY INFECTION

The removal of the transverse processes aids greatly in the obliteration of that portion of the pleural cavity lying in the vertebral gutter along the sides of the vertebral bodies. This is the space where residual sinuses are apt to occur.

The first rib must be removed anteriorly beyond the subclavian groove and if possible it is well to divide it beyond the attachment of the scalenus anticus muscle. This allows the obliteration of the extreme dome of the pleural cavity. It is important to remove the second and third ribs up to their attachments to the costal cartilages. These two ribs are the ones which hold out the upper portion of the pleural space and anything short of their complete removal will not serve the purpose. The full effect of the sinking in of the scapula is not obtained until a portion of the sixth rib has been removed, because the lower angle of the scapula rides on this rib and is held out by it. The fourth, fifth and sixth ribs are divided at the anterior axillary line or anterior to it, depending on whether the lung is partially or completely collapsed, and whether the right or left side is involved. As a rule more extensive portions of these ribs are removed on the right than on the left side. The remaining ribs are removed at their point of attachment to the diaphragm.

The success of this procedure depends on two important factors, both of which make this distinctly advantageous to the operations which attempt obliteration of the pleural cavity by removing the pleura and intercostal muscles first; two layers of tuberculous pleura when brought in contact with one another adhere even in the presence of pyogenic organisms provided the exudate is not allowed to accumulate. This must be prevented by adequate drainage and the removal of any air either by frequent aspiration or the introduction of a catheter. Second, the operative wounds heal by primary union because the procedure is undertaken in normal healthy tissue.

The foregoing procedure may be modified in cases having particularly extensive cavities or extremely thick or calcareous pleura. In these the sixth rib and transverse process are removed subperiosteally and then the fifth, fourth, third and second ribs are denuded of periosteum but left *in situ*. A portion of the first rib is then removed. The space between the denuded ribs on one side and the intercostal muscles and periosteum on the other is then firmly packed with gauze, pressing the parietal pleura, the intercostal muscles and the periosteum over toward the mediastinum. The skin and muscular flap is then sutured in place. After ten days the wound is reopened, the ribs removed and the gauze pack is taken out. This leaves two raw surfaces covered by fresh granulations. The wound is then closed without drainage. The advantage of this procedure is an immediate obliteration of the pleural space following the first step. A calcareous or very thick pleura is mechanically forced into a position where the two pleural surfaces are held in contact and the possibilities of failure due to a residual cavity are diminished.

In the cases in which open drainage has been established there will always remain a sinus or comparatively small cavity on the diaphragm. This may be closed in several ways. Paralysis of the diaphragm following division of the

phrenic will at times aid in obliterating these cavities situated on the diaphragm. We reserve the division of the phrenic until after the completion of the thoracoplasty believing that contraction of the diaphragm is an aid in drawing the anterior ends of the divided seventh to tenth ribs inward and that it is a disadvantage to have the abdominal viscera push upward toward the thorax and hold these rib ends outward. This is especially true on the right side where the liver serves as a compact solid organ.

The frequent and repeated cauterization of persistent pleural sinuses with 95 per cent. carbolic acid will often close them. One must be meticulous, however, to cauterize the extreme depths of the sinus, otherwise the superficial portion will heal and a residual abscess reform. In a few instances where the cavity has been small or where a narrow tract has persisted we have laid these open and treated them as open granulating wounds.

SKELETAL MUSCLE SARCOMA

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TUMORS involving the skeletal muscles, while not of frequent occurrence, are by no means rare. Of these the two most common types are hæmangioma and sarcomata. The former have been most adequately studied, and have been reported in large numbers.^{6,11,16} Hence they have been considered the more common. It has been the author's experience that the sarcomata are of equal if not greater frequency. Since 1913, when Küttner and Landois¹³ reported 130 cases of skeletal muscle sarcomata culled from the literature, and added sixteen of their own, there has been no large series described. In reviewing the case records of recent years at the Hospital for Joint Diseases and Mt. Sinai Hospital, we were able to collect thirty-five instances of skeletal muscle tumor. Table I shows their histogenetic distribution.

TABLE I

Hæmangioma	11
Sarcoma	
Fibrosarcoma	13
Neurofibrosarcoma	4
Myxosarcoma	1
Myosarcoma	1
Chondrosarcoma	1
	—
	20
Fibroma	1
Neurofibroma (excluding von Recklinghausen's	
multiple neurofibromatosis)	2
Carcinoma	1
	—
Total	35

Of thirty-five tumors involving the skeletal muscles, twenty, or 57 per cent., were sarcomata, and eleven, or 31 per cent., were hæmangiomata. Of the sarcomata, the greater number, by far, were classified as fibrosarcomata. In Ewing,⁷ the same observation is made, without statistics, regarding the preponderance of fibrosarcomata among skeletal muscle sarcomata. The differentiation of neurofibrosarcoma from fibrosarcoma is difficult, and in some cases impossible. The former is apt to be encapsulated or, at least, well circumscribed; the latter more likely to be diffuse or less well demarcated. In our series neurofibrosarcomata were second in frequency (four cases). In addition to the sarcomata classified in Table I, two other types have been reported. Osteogenic sarcoma occasionally invades the adjacent muscle, especially at sites where muscle origin or insertion is directly in contact with

TABLE II
Muscle Sarcomata—20 Cases

Classification	Muscle	Age	Sex	Duration	Symptoms	Therapy	Remarks
1. Fibrosarcoma	Hamstring m. group	60	F.	5 mos.	Soft tumor, non-mobile, grape-fruit size. Painful only during last few weeks	Excised	Tumor was a recurrent growth; X-ray treatment had been advised after first operation, but was refused
2. Fibrosarcoma	Rectus abdominis m.	27	F.		Sausage-shaped tumor, 10 x 5 cms., firm, mobile. No pain or tenderness	Excised with resection of muscle	Desmoid type of tumor; histological study proved it to be sarcomatous
3. Fibrosarcoma	Internal oblique m.	27	F.	1 yr.	Orange-sized, firm tumor, appearing one month after an attack of acute right lower quadrant pain. No pain or tenderness after appearance of tumor	Excised with resection of muscle	This tumor, although a "desmoid," grew rapidly
4. Fibrosarcoma	Flexor muscles of forearm	42	M.		Slowly growing tumor; no pain or tenderness at any time	Excised	
5. Fibrosarcoma	Extensor communis digitorum m.	13	F.	2½ yrs.	Slowly growing tumor; firm; no pain or tenderness at any time	Excised	Apparently originating from visceral aspect of muscle sheath, involving muscle tissue directly, without perforating the sheath
6. Fibrosarcoma	Hamstring m. group	31	F.		Large tumor, surrounding thigh. Painful; firm	No treatment	This was a third recurrence, following two surgical excisions
7. Fibrosarcoma	Biceps femoris m.	60	F.	5 mos.	Orange-sized; firm tumor; painless until 3 wks. before admission; then felt a dull ache	Excised	
8. Fibrosarcoma	Triceps brachialis m.	24	F.	2 mos.	Slowly growing tumor. No pain or tenderness at any time	Excised	
9. Fibrosarcoma	Rectus abdominis m.	22	F.	3 mos.	Firm tumor, sausage-shaped. No pain or tenderness at any time	Excised	

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10. Fibrosarcoma	Trapezius m.	35	F.	1 yr.	Pain in occiput and upper spine 3 mos. At end of this time, a firm, slowly growing tumor appeared in trapezius area. No further pain until one month before admission. Then lancinating pain radiating up occiput and down arm	Excised	Note presence of distinct radiation of pain along trunk distribution. May have been an intramuscular neurofibrosarcoma
11. Fibrosarcoma	Rectus abdominalis m.	31	F.		Lemon-sized tumor. No pain or tenderness at any time	Excised	
12. Fibrosarcoma	Semitendinosus and semimembranosus mm.	10	F.	6 wks.	Firm tumor. No pain or tenderness at any time	Excised	Encapsulated; fixed to visceral layer of muscle sheaths
13. Fibrosarcoma	Rectus abdominalis m.	28	F.	7 mos.	Firm tumor. No pain or tenderness at any time	Excised	Tumor first noted during abdominal enlargement of pregnancy
14. Neurofibrosarcoma	Flexor muscles of forearm	26	M.	9 yrs.	Firm, mobile tumor. Moderate pain	Excised	This was a second operation for recurrence
15. Neurofibrosarcoma	Extensor muscles of forearm	29	M.	9 yrs.	Walnut-sized tumor; firm; mobile. Tender. Pain for 5 days before admission, radiating down ulnar nerve	Excised	This was a second operation for recurrence
16. Neurofibrosarcoma	Peroneus longus m.	26	M.	Since childhood	Walnut-sized tumor; progressive growth following trauma about 1 yr. before admission. Tender. Pain persisted following trauma	Excised	Tumor was encapsulated
17. Neurofibrosarcoma	Gastrocnemius m.	42	F.	7 yrs.	Tumor present 7 yrs.; no change in size since first noted, until several months before admission began to grow rapidly. No known trauma. Tumor pulsation on palpation	Excised	Tumor was encapsulated
18. Myxosarcoma	Flexor muscle group of forearm	52	F.	3 yrs.	Soft tumor; tender; painful	Excised	
19. Fibrochondrosarcoma	Palmaris brevis m.	23	F.		Tumor present on hypothenar eminence. Painful. No known trauma.		
20. Myxosarcoma (probably Fibrosarcoma)	Vastus lateralis			3 mos.	Grapefruit-sized tumor; growth; painful when walking. Invaded surrounding structures	Excised	Although classified originally as myxosarcoma, this tumor had clinical characteristics of a fibrosarcoma. Slides not now available

bone; that is, at points of osteoperiosteo-muscle conjunction. However, osteogenic sarcomata may at times invade adjacent muscle by perforating both periosteum and muscle sheath.^{12,9} Rhabdomyosarcomata have been reported by Cohen,⁵ Wolbach,²⁰ and Muller.¹⁷ The diagnosis of rhabdomyoma or its complexes has been doubted by several pathologists.^{7,19,13} Its true nature brings up a problem in histogenesis extraneous to the present purpose. Briefly, the term muscle tumor designates a neoplasm involving the muscle body. It must not necessarily be composed of muscle fibres; in fact, it rarely, if ever, is. In certain sarcomata of skeletal muscle, the muscle cells play a secondary part, in others they are concerned only spacially. In the presence of a rapidly growing sarcoma the muscle cells may form part of the stroma due to the infiltrative type of growth characteristic of these tumors.¹⁴ Whether a so-called rhabdomyosarcoma is a true derivative of muscle cells, or a sarcoma or teratoma in which such cells are fortuitously included, is the crux of this problem.

Sarcoma may involve the skeletal muscle in several ways. First, the tissue of origin may be intramuscular; that is, sarcolemma, perimysium, interfascicular connective tissue, the visceral layer of muscle sheath, perineural or perivascular connective-tissue sheaths. Secondly, the tumor may infiltrate the muscle body from a source outside, but in direct contact with it, such as, for example, a fibrosarcoma of periosteal or intermuscular fascial origin. This infiltration by direct contact is also the source of intramuscular osteo- and chondrosarcomata. Thirdly, the muscle body may be involved by a metastatic process as occurs when a large secondary fibrosarcoma appears in the muscles and surrounding soft tissues of the thigh following the removal of a supposedly benign fibroma from the plantar surface of the foot.⁴

Age.—The statement is repeatedly made that sarcoma occurs most commonly in youth or the young adult. However, fibrosarcoma of the extremities may occur at any age and, in fact, was found to be most frequent in the fourth and fifth decades.³ The ages of the twenty cases herein listed range from ten to sixty, appearing with maximum frequency between twenty and forty. The age groups for fibrosarcoma and neurofibrosarcoma were similar. This is in marked contrast to a characteristic age of origin for intramuscular hæmangiomata. The latter, in all probability, invariably a congenital lesion,^{6,11} is at least usually first noted in early childhood.

Duration.—Of nine cases of fibrosarcomata in which the duration of tumor was noted, in all but one it was a year or less. In one case it was two and a half years. (Comparable figures not given by Küttner and Landois.) In four cases of neurofibrosarcomata the duration was from seven to twenty-six years. The long duration of a static neurofibrosarcoma followed by a rather brief period of rapid growth, often, but not always after trauma, is characteristic of these tumors of nerve-sheath derivation.⁴ Likewise, it is not unusual for a fibrosarcoma of the anterior abdominal wall to be discovered only at the time of pregnancy when distention of the musculature brings a hitherto unnoticed sausage-shaped tumor into prominence.

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Although a muscle tumor of relatively short duration suggests a diagnosis of fibrosarcoma, one of longer duration does not necessarily signify benignity. In the presence of a neoplasm which has existed from childhood, hæmangioma must be considered; in one of long duration having an onset later in life, a neurofibrosarcoma is a probable diagnosis.

Location.—The sites of predilection for intramuscular sarcomata are the thigh, abdominal wall, and forearm, sixteen of the twenty cases occurring in these regions with approximately equal frequency. Two of the neurofibrosarcomata occurred in the leg and two in the forearm. Fibrosarcomata occurring in the abdominal musculature have long been known under an old clinical name, "desmoid," *i.e.*, fibrous tumor. These usually involve the rectus abdominis of either side.

Pain.—Pain is rarely an early symptom. In most instances, although a minor ache may have been present at recurring intervals, actual pain is either never experienced, or appears only when the tumor is well advanced in size. When present, it is apt to be described as "rheumatic." Unlike bone sarcoma the pain is seldom constant. The presence of pain radiating down a specified nerve distribution is suggestive of, but not pathognomonic for a neurogenic tumor or neurofibrosarcoma.^{3,4} Any muscle tumor which in its growth involves a nerve spacially will produce similar radiating pain. Muscle sarcomata may or may not be tender. Usually they are not. However, occasionally one finds some moderate pain present for a while after a slight trauma.

The absence or inconsequentiality of pain in these tumors explains the rarity of early diagnosis. Situated as they are in deep resilient structures, covered by soft subcutaneous fat, the tumor of itself draws no attention until its presence is visibly perceptible. This occurs only after it has attained a considerable size. Occasionally, however, palpation during the course of a careful examination may discover it before a visible tumor appears, but, even so, it must have reached a fairly large size before becoming palpable through the overlying tissues.

Mobility.—Primary muscle tumors in general have a characteristic mobility, especially when felt at a stage in their growth before adjacent tissues are involved. They are deep-seated; the skin and superficial tissues are mobile in reference to the tumor; the tumor is mobile in reference to underlying bone; and it can be felt to change position during contraction or elongation of the muscle. The amplitude of change in position during muscle activity diminishes as its situation approaches the functional mid-point of the muscle, and increases as it is distant from that point. Until the size of the intramuscular tumor is such that it mechanically interferes with the excursion of the muscle, or until in its growth it perforates the muscle sheath and attaches to adjacent fixed tissues, there is no perceptible disturbance in muscle function. This, of course, does not apply to muscle tumors of the second category (*v.s.*), where the muscle has been involved by infiltration

from adjacent tissues. In these latter cases mobility is either entirely restricted or is palpable as a mass waving on its pedicle.

Size.—In size, skeletal muscle sarcomata when first seen range from that of a pigeon's egg to a large grapefruit. The latter are not infrequently encountered in the thigh. When occurring in the abdominal wall they usually resemble a large frankfurter sausage in size and shape.

Consistency.—These tumors are characteristically firm, occasionally to the point of simulating bony hardness. A soft muscle tumor suggests but cannot define a hæmangioma, since a myxosarcoma may be quite limpid to the touch.

Gross pathology.—A knowledge of the growth characteristics of sarcomata is essential to a proper conception of therapeutic approach. These neoplasms grow not by invasion, as does carcinoma with its gross displacement of the recipient tissue, but rather by infiltration, an insidious penetration of the connective-tissue framework of the area involved. Hence a definite line of demarcation never becomes apparent. A gross mass may be seen where the sarcomatous tissue is dense, but the borders of this mass will be fringed with long strands of neoplastic fibrils extending into the adjacent connective-tissue pathways. It is only in the neurofibrosarcomata that encapsulation is sometimes found. In these tumors there seems to be some factor (a problem, incidentally, as yet unsolved) which confines the local growth, though not the metastatic, to connective tissue of nerve-sheath origin.

The malignancy of sarcoma varies as does that of carcinoma. Experience teaches, for example, that the desmoid of the anterior abdominal wall is almost always of low malignancy. When metastasis occurs in sarcoma it takes place directly through the blood-vessels rather than through the lymphatics.⁷ Hence, enlargement of the proximal lymph-nodes does not occur in sarcomata of the extremities as it invariably does in carcinoma.³ In sarcoma, metastasis is apt to appear first in the lungs, arriving through the blood-stream without intermediate rest points. Thus the potential danger to life is considerable even in the early stages of muscle sarcoma.

Biopsy.—The value of biopsy in skeletal muscle sarcoma is the same as that for other sarcoma. The inherent sources of error must be weighed against the invaluable advantages of a true diagnosis when it is obtained. This problem is also extraneous to the present study. However, it may be here stated that the purpose of this series of studies by the present author, as of similar studies by other surgeons, is to so define and describe the clinical aspect of many tumors as to make diagnosis possible, or, at least, reasonably certain, on the basis of clinical history and examination. In this way, the X-ray and biopsy will assume their proper functions as methods of laboratory confirmation in well-indicated cases.

Therapy.—The treatment of primary intramuscular fibrosarcoma or myxosarcoma is excision of the tumor with the surrounding muscle tissue as extensively as is feasible; if necessary, even to the resection of the entire

SKELETAL MUSCLE SARCOMA

muscle. Close scrutiny must be maintained for evidence of involvement of nearby fascial layers. Following this there should be given a prolonged course of deep radiation. This is advocated with full appreciation for the fact that tumors of connective-tissue origin stand well down in the series of radiosensitivity. However, the danger to life is such that any additional factor of safety beyond surgical excision should be added to the treatment. Radiation properly applied over a sufficiently prolonged period of time may in some instances be a life-saving device. In the presence of the secondary type of muscle sarcoma, the excision must of course include a wider area. In such cases involving the muscles of extremities, amputation is the treatment of choice.

Neurofibrosarcomata are among the most radioresistant of all sarcomata. It is doubtful whether radiation ever affects them. On the other hand, the complete excision of intramuscular neurofibrosarcomata is often possible due to the presence of a well-encapsulated mass (v.s.). It is our opinion that, with present knowledge, nothing can be done in these cases to avoid metastasis other than amputation. The success of therapy in skeletal muscle sarcomata depends upon (1) the grade of malignancy of the tumor at the time of treatment; (2) early recognition and differentiation, and (3) the completeness of excision. These factors are given according to their degrees of importance. Radiation, while always to be advocated, is still of doubtful efficacy in determining the outcome of any given case.

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LOCALIZED CHRONIC ULCERATIVE ILEITIS

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FOR the last twenty-five years and during the last decade particularly, there have been sporadic reports in the literature of cases loosely classified under the heading granulomata of the intestine.

Until 1909, when Heinrich Braun referred to a case previously reported by him and collected reports on several other cases, the condition was practically disregarded in both Europe and America. Probably many cases of intestinal granulomata had been seen prior to this time; in fact some had been reported. But the majority of such cases were relegated to a pathological discard under such headings as tuberculomata, malignancies and the granulomata of syphilis, and their true nature never questioned or studied.

In 1909, Braun, pointing out the importance and difficulty of differentiating these intestinal granulomata from neoplasms of the intestine, collected six cases (including two of his own) of this type. The granulomata were situated at various places in the large intestine and the majority caused obstructive symptoms. In most cases a mass could be felt. In none of the cases was the etiology apparent. In 1920, Tietze⁶ reviewed all literature on this type of case and added to it several cases of his own. Moschcowitz and Wilensky,⁵ in 1923, reported four cases, in one of which the terminal ileum was involved. In 1925, Coffen⁴ added a case to the literature and cited previous cases as reported by Braun, Moschcowitz and Wilensky. In 1931, Mock³ described ten cases of granulomata and, in 1932, Golob² reported another case, discussed the subject further, and made some suggestions as to its etiology.

The etiology of the condition is exceedingly obscure. Many cases of granulomata are found apparently arising around a foreign body. Mock believes that they are usually due to a low-grade infection. Golob cited a case in which he believed the presence of a duodenal ulcer with its "constantly irritating influence over the ileocaecal region" might have been a predisposing factor.

Until 1932 no attempt had been made to differentiate a specific entity from the types of cases which had previously been reported. They were simply called intestinal granulomata, and they were found throughout the large intestine, in the omentum and occasionally involving the terminal ileum and proximal cæcum. In 1932 Crohn and his co-workers¹ isolated from this mass of heterogeneous granulomata a specific entity which they called "regional ileitis." In a review of the work by Tietze and Mock they were unable to find a report of a case which approached the picture which they had discussed. In one case reported by Moschcowitz and Wilensky, however,

there was a close resemblance to the type of case they classified as regional ileitis.

Regional ileitis, as described by Crohn *et al.*, is a disease which clinically suggests ulcerative colitis. It is characterized by fever, diarrhoea, and emaciation and eventually leads to intestinal obstruction requiring surgical interference. In all cases a mass is present in the lower right iliac fossa. In all cases the terminal ileum is alone involved. The process begins at the ileocaecal valve and extends upward, involving the ileum usually for a distance of from 20 to 30 centimetres. Often there are fistulae leading to adjacent segments of the colon and occasionally to the anterior abdominal wall. The etiology of the condition is unknown.

Characteristically, the pathology is as follows: The inflammatory process begins at the ileocaecal valve and usually involves 20 to 25 centimetres of the distal ileum. It is most pronounced at the valve, the process becoming less severe and gradually shading off into normal intestine, proximally. The submucosal, and to a less extent the muscular, layers of the bowel show hyperplastic and inflammatory changes. The walls are thickened, the lumen made smaller. The adjacent mesentery is greatly thickened and fibrotic.

The formation of fistulous tracts into adjacent loops of bowel (sigmoid, caecum, colon) is common. These fistulae are preceded by slow perforations giving rise to walled-off abscesses which, if drained, result in intractable faecal fistulae.

Microscopically the picture is one of non-specific inflammation and proliferation. In many cases the mucous membrane is destroyed and often replaced by an atrophic layer of epithelium. Giant cells are occasionally seen. They are interpreted by Crohn to be due to a foreign-body reaction to minute particles of vegetable material entrapped in lymphatics during the process of healing.

In half of the cases reported by Crohn, previous appendectomy had been done. It is pointed out that in cases in which no appendectomy had been done, the walls of the appendix showed inflammatory change but the mucosa was normal.

The disease is limited to young adults. Males are more often affected. The disease lasts over months or years. Diarrhoea, fever, loss of weight and anaemia are almost constant features. The temperature is usually intermittent with periods of remission. It rarely goes higher than 103° F. The diarrhoea is the chief complaint. Two to four stools a day of varying consistency, but always containing mucus and often blood and pus, are usually the case. Tenesmus is always lacking.

The authors state that perirectal abscesses, condylomata and peri-anal fissures are never found since the colon and rectum are not involved.

Vomiting, accompanied by pain and visible peristalsis, is present in the stenotic cases. The pain is dull and cramp-like and may be fairly general in the lower abdomen or localized in the right lower quadrant. Occult blood is found in the stools. The white count is usually normal but may be slightly elevated.

The authors list characteristic findings in the physical examination as follows:

(1) A mass in the right lower quadrant. (2) Evidence of fistula formation. (3) Emaciation and anaemia. (4) Evidences of previous appendectomy. (5) Evidences of intestinal obstruction.

The disease may be divided into four types, according to Crohn. In one type the disease simulates acute appendicitis. There are signs of acute intra-abdominal inflammation with an elevation of temperature and leucocyte count, tenderness in the lower right quadrant and occasionally a mass in this area. If operation is done at this time, the terminal ileum is greatly thickened, red, blotchy and surrounded by oedematous tissues. Peri-appendicitis is present, and occasionally one finds an abscess. In another type symptoms of ulcerative enteritis with colicky abdominal pains, diarrhoea, and elevated

CHRONIC ULCERATIVE ILEITIS

temperature predominate. Loss of weight, anæmia, and general weakness are characteristic of the later phases of this type. Clinically it passes slowly into the stenotic type of the disease. In the stenotic type, which is the most common, the symptoms are those of small bowel obstruction of varying degree. Cramps, borborygmus, occasional vomiting, and constipation may be present. In the last type the outstanding feature is the presence of persistent fecal fistulæ. These fistulæ follow an attempt to drain what are thought to be appendiceal abscesses. Fistulæ, it is pointed out, may develop after the original drainage wound has been healed for several months.

In the röntgenographical examination two observations are of value in the diagnosis. Because of the clinical resemblance of this disease to ulcerative colitis, a barium enema is usually first given. This examination is negative because the disease stops at the ileocæcal valve. A barium meal, however, usually denotes a fluid level in the terminal ileum and delayed motivity in this region. In the stenotic type the delay is pronounced.

Regional ileitis is to be differentiated from ulcerative colitis, ileocæcal tuberculosis, fibroplastic appendicitis, carcinoma of the terminal ileum, Hodgkin's disease, actinomycosis, sarcoma, intestinal or mesenteric tuberculosis and non-specific proctitis.

The treatment of the condition is primarily surgical. Medical treatment is palliative and supportive. Resection of the diseased segment leads to a cure of the condition in all patients surviving the operation.

CASE REPORT.—A man aged thirty-nine was admitted to the Billings Hospital September 19, 1932, on the service of Doctor Palmer complaining of diarrhœa of four years' standing, intermittent abdominal cramps, loss of 49 pounds in four years. Until five years ago he had been entirely well. He then developed a diarrhœa and had as many as fifteen or sixteen thin, watery stools daily. He averaged from six to eight stools daily. This diarrhœa persisted intermittently, with occasional remissions lasting from several days to a month, until his entrance to the hospital. Occasionally there was tenesmus associated with this diarrhœa, and though there was mucus in nearly all the stools, no blood or pus was ever seen. During the remissions, the stools were usually of a normal consistency; however, there were always at least two movements daily. The diarrhœa occurred without relation to the type of food eaten or to the activity of the patient. Associated with this diarrhœa, were abdominal cramps which were partially relieved by defecation and enemata, and an almost constant, dull, aching pain in the lower abdomen, which frequently radiated to the right loin.

The patient's family physician sent him to a nearby hospital for observation. While there gastro-intestinal X-rays were taken, gastric analysis made and stools examined. After three days the patient was discharged, told that there was nothing organically wrong with him, and put on a low-residue diet of milk, cereals, *etc.* He adhered to this diet for six weeks with no relief.

In about August of 1928 he went to a clinic where, after eight days of observation, he was told that he had a fissure in ano. He was advised to rest in the country, eat a normal diet and take retention enemata of warm olive oil twice daily. This advice was followed, the patient remaining in the country for one month. At the end of this time he had experienced no relief. He sought the advice of another doctor in Chicago and remained under his care for three months. During this time the treatment was purely dietary with the exception of enemata given night and morning. After three months of this treatment, during which the patient felt somewhat relieved, he developed an ischiorectal abscess. This was opened and the physician stated that it was due to a fistula and that it would heal spontaneously. The drainage persisted for three months and finally the patient went for treatment to a sanitarium which specialized in rectal disease. During all this time the diarrhœa and abdominal pains persisted intermittently. After eight operations the patient was discharged as cured so far as the fistula was concerned. He went home for about two months. When he left the hospital he felt much better and was having only occasional cramps and diarrhœa. About two months

later, however, the diarrhoea and pains grew worse and he again went to a clinic for examination and treatment.

Before entering the clinic he noticed that he felt feverish. During his five weeks' stay he ran a temperature of from 98.6° to 103° F. The fever persisted until about ten days before his dismissal. While in the clinic the patient developed frequency (about every thirty minutes), burning, and nocturia. He was cystoscoped and told that his urinary symptoms were due to mechanical causes. The urinary difficulties lasted until about three weeks after his dismissal from the hospital.

The patient was told on entering the clinic that a mass could be felt in his rectum and that it could be seen in X-ray examination. Colonic irrigations were instigated and diathermy treatments given. The patient states that as a result of this treatment he felt much better. The diarrhoea and pain disappeared and he gained in weight. He was discharged from the hospital and told to continue the diathermy and irrigations. This he did until January, 1932. After leaving the clinic the diarrhoea and abdominal pain recurred but were neither so severe nor so frequent as before. The patient discontinued the diathermy. After a brief period, pain and diarrhoea became more severe. He again tried diathermy, this time with no relief. He entered this hospital in August, 1932, for observation and treatment.

His past history, with the exception of that which has already been given, was largely irrelevant. His best weight five years ago was 210 pounds. One year before entering the hospital he weighed 143 pounds. He had lost 20 pounds in the last six weeks.

It was brought out in the history that his attacks of pain were much worse at night and that a bowel movement only partially relieved them. Associated with the abdominal pain there was a right lumbar pain, which, though less severe than the cramps, lasted long after they had ceased. In addition to the complaints of diarrhoea, pain, and progressive weakness and loss of weight, the patient stated that for several months he had had frequency of six to eight times a day, nocturia of two times, and dribbling and incontinence which had been growing progressively worse for the last six weeks.

He was a fairly well-developed, fairly well-nourished white male who looked at least five years older than his stated age, and who was not acutely ill. The physical examination was essentially negative except for the following findings:

Heart.—There was a soft systolic murmur over the aortic area which was transmitted up the neck. The heart was otherwise normal.

Abdomen.—The abdominal musculature was well developed and somewhat spastic. There was some generalized tenderness all over the lower abdomen. The entire abdomen was tympanitic. No fluid was found on percussion nor were there any areas of definite dullness. The bowel was distended and vigorous peristalsis was seen to occur at five-minute intervals.

During a typical attack of pain the spasticity of the muscles of the lower right quadrant increased enormously and a definite swelling could be seen in this area. At the same time visible peristalsis occurred and then, accompanied by much rumbling and gurgling, the mass diminished in size and, together with the spasticity of the overlying musculature, disappeared.

Rectal examination revealed the scars of previous operations. At the upper edge of the prostate a large, irregular mass was felt which did not seem to involve the rectal wall. The mass was not ballotable.

Proctoscopic examination revealed a sudden narrowing of the lumen of the rectum at about 12 centimetres as if from pressure from without. The proctoscope could not be manipulated beyond this point.

Laboratory Findings.—Blood: hæmoglobin 92 per cent. Sahli; red blood cells 4,490,000; white blood cells 6,500; differential polymorphonuclears 57; large leucocytes 2; small leucocytes 34; mononuclears 4; eosinophiles 4; basophiles 0. Blood Wassermann and Kahn negative. Urine negative except for 5 to 10 white blood cells per high powered

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field on two out of six examinations. Stools negative for blood and mucus on all examinations.

The patient remained on the medical service for nine days after his entrance into the hospital. During this time a gastro-intestinal X-ray was made.

X-ray Report.—Colon fluoroscopy August 22, 1932. With enema the colon fills easily and completely from the rectum to the tip of the cæcum and after vigorous manipulation through the abdominal wall the barium enters the ileum in a fine stream. The appendix is not seen.

Stomach fluoroscopy August 23, 1932. Barium given yesterday is scattered throughout colon. Oral barium is taken without difficulty. The œsophagus and stomach are entirely normal except that the latter is in high transverse position with a posterior bulb. (Figs. 1 and 2.)



FIG. 1.



FIG. 2.

FIG. 1.—Barium enema five days before operation showing sharp retention of barium at ileocecal valve.
FIG. 2.—Barium meal four days before operation, twenty-three hours after ingestion. Note enormous dilation of terminal coils of ileum with typical stepladder arrangement. Stoppage of barium at a point near the ileocecal valve.

Serial No. 3. Abdomen: Patient prone, six hours after the ingestion of the oral barium. The stomach is empty. The coils of small intestine contain most of the barium and appear enormously dilated. Small amounts are also seen in the colon.

Serial No. 4. Twenty-three hours after ingestion of barium. There is still considerable retention of the barium within the small intestine. Barium is also seen in the cæcum, transverse and descending colon.

Serial No. 5. Approximately forty-eight hours after ingestion of oral barium. The barium has now almost completely left the small intestine and is within the colon. Obviously there is delayed emptying time of the small intestine with marked dilation of it. We have not been able to demonstrate a mass within the gastro-intestinal tract though we can infer the presence of something within the abdomen causing a partial obstruction to the passage of the barium meal. The impression resulting was of a mass (?) within the abdomen causing delayed motility of barium through the small intestine.

While on the medical service the patient was given tincture of belladonna and

deodorized tincture of opium in an effort to relieve his symptoms. Further study of the case was interrupted about a week after the patient's entrance into the hospital by the rapid onset of alarming signs of acute intestinal obstruction which seemed to be located at the ileocaecal valve which had previously been shown to be strictured.

Operation August 27, 1932. Ethylene anaesthesia. Dr. Andrews. Through a right-rectus incision an enormous mass of matted, indurated bowel was discovered in the cul-de-sac. This was freed by finger dissection and the pocket from which it was taken packed with gauze. The mass was then delivered outside the abdomen and found to be composed of terminal ileum. The appendix and caecum were grossly normal. The ileum, together with its mesentery, was enormously thickened for a distance of about 8 inches above the ileocaecal valve. The ileum above this mass was markedly hypertrophied due to the obstruction. Scattered throughout the mesentery were large, indurated glands, some of which fluctuated. Exploration of the abdominal cavity revealed no signs of tuberculosis. It was therefore decided that the intestinal pathology was due to a low-grade pyogenic infection in the terminal ileum.



FIG. 3.—Photograph of excised specimen. Note enormous thickness of walls of terminal ileum and normal appendix and caecum at bottom.

A Mikulicz exteriorization was made and a portion of the caecum, together with the indurated ileum, was brought outside the abdominal wall and sutured in place. The abdominal wall was then partially closed around the loop of exteriorized intestine.

The next day, under nitrous-oxide anaesthesia, the terminal portion of the ileum and proximal portion of the caecum were amputated with the actual cautery and a right-angle clamp applied to the spur. The walls of the ileum measured 3 to 4 centimetres in thickness and the lumen was about 1 centimetre in diameter. The wound was partially closed with interrupted silk sutures.

Pathological Report.—Gross.—The specimen is that of terminal ileum, appendix and proximal caecum. The ileum is enormously hypertrophied and indurated. The attached mesentery is markedly thickened, indurated and hyperaemic and contains many hyperplastic lymph-nodes which display a reddish-gray pulp on cut section. The walls of the appendix and caecum are slightly thickened.

On cut section, the walls of the ileum, particularly at its distal end, are seen to be enormously thickened and fibrotic (Fig. 3), the lumen being encroached upon. Near the ileocaecal junction the walls measure 3 to 4 centimetres in thickness while the lumen

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is only 1 centimetre in diameter. The hyperplasia of the walls becomes gradually less marked proximally, and at about 25 centimetres from the ileocaecal junction the ileum is apparently normal. At various places near the ileocaecal junction, the mucosa is denuded and exhibits shallow serpiginous ulcers with low margins.

Microscopical.—The walls of the ileum are markedly thickened and oedematous. The mucosa is lacking in some areas with the formation of shallow ulcers with overhanging edges. There is a marked diffuse and focal lymphocytic infiltration of the submucosal layers, the normal lymph follicles are hyperplastic and the vessels are engorged. (Fig. 4.) The muscular layer is markedly hypertrophied and exhibits diffuse leucocytic infiltration. The serosa is covered by a thin layer of leucocytes and degenerating fibrin. No granulomas are present in any of the sections.

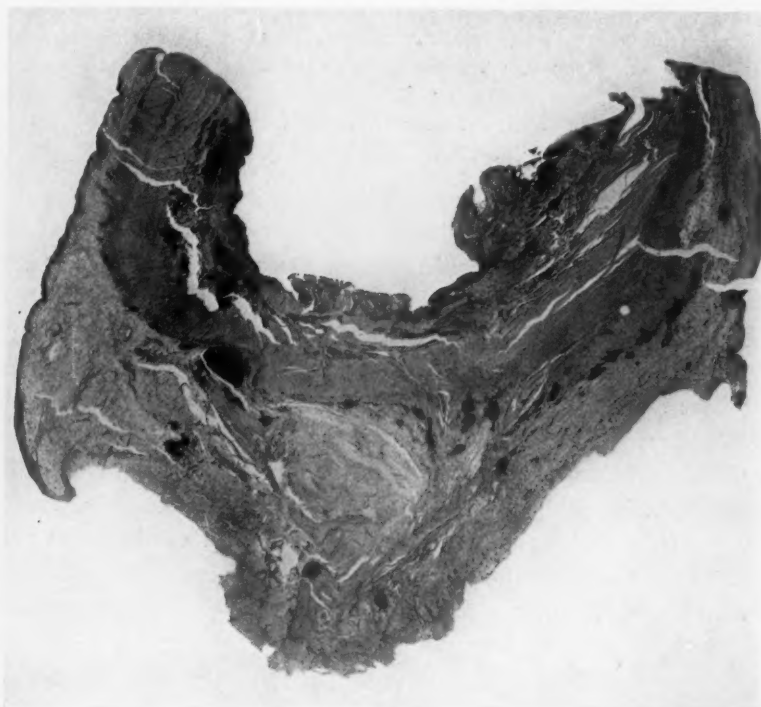


FIG. 4.—2X magnification of section through inflamed area, showing numerous ulcerations, hypertrophy and oedema of outer layers and marked focal collections of lymphocytes.

Sections from the appendix are essentially negative except for a thin layer of fibrino-purulent exudate over the serosa.

Following this operation the patient made a fairly rapid convalescence, his urinary symptoms entirely disappeared, and he regained his appetite and was relieved of all pain. The diarrhoea, however, persisted, probably due to the fact that all the irritated portion of the ileum had not been removed at the operation. The liquid ileostomy drainage was alkaline (Ph 8) and had a marked digestive action on the wound edges, which became excoriated and extremely tender. In an effort to neutralize this faecal drainage, continuous irrigation with 1/100 N hydrochloric acid was instigated. This was combined with continuous suction designed to carry away excess faecal material and irrigating solution. The condition of the wound materially improved under this treatment. In addition to this persistent diarrhoea from the ileostomy there was marked infection

of the wound and considerable purulent drainage, possibly due to the breaking down of the mesenteric glands seen at operation.

It was not considered safe to attempt a closure of the ileostomy in the face of such marked infection.

On the twenty-fourth of October, about a month after the original operation, the patient, who had been up in a wheel chair for two days, suddenly developed all the signs of an acute intestinal obstruction with vomiting, distention, and marked visible persistalsis.

On the following day under ethylene anaesthesia, laparotomy through the old operative wound was done and revealed a fibrous band constricting the ileum just proximal to the ileostomy opening. This was divided, allowing material which had been dammed back in the ileum to well up in the field. A catheter was sutured into the ileostomy opening and the gut closed around it. The wound was then closed around the tube in layers.

Drainage from the tube persisted for about six days. The tube was then withdrawn and the wound strapped with adhesive tape. Healing took place almost by first intention. The patient was discharged November 20, 1932. At that time there was a very small amount of purulent drainage from the wound. There had been no faecal drainage since the removal of the tube.

Discussion.—This case seems to us to belong in the category described by Crohn and called by him regional ileitis. It belongs in the type which they designate as stenotic. It differs from their cases in several respects. None of their cases had had rectal fissures or fistulae, as had our case. Tenesmus was not one of the characteristic features of the cases they reported. Furthermore, the formation of fistulae from the affected gut to adjacent colon or sigmoid was an important finding in their cases which was not present in ours. We feel that the differences between our case and the cases they described may be attributed to the fact that in our case the affected ileum was bound down in cul-de-sac. Since the history of the disease dates back well before the appearance of the rectal abscess, it may be assumed that the ileitis preceded the abscess formation. If this is true, it would seem entirely possible that the presence of this inflammation in the cul-de-sac could easily give rise to an abscess, which, if drained, would cause a persistent rectal fistula, which, as in their cases of abdominal fistulae, was very difficult to cure because of the underlying inflammatory mass.

Furthermore, the occasional tenesmus which our case had could also be attributed to the proximity of the ileitis to, and its consequent irritation of, the rectum.

The urinary symptoms, so outstanding a feature of our case, were certainly due to pressure and irritation of the bladder by the mass in the cul-de-sac.

Another feature of our case which was due to the position of the involved ileum is the fact that it might be easily confused with non-specific proctitis. In this condition a brawny, indurated mass outside the rectal wall is one of the outstanding features.

The absence of secondary anaemia in our case can possibly be explained by the remissions which our patient had had repeatedly during the course of the disease.

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In all other respects the picture is certainly typical of the regional ileitis described by Crohn.

NOTE.—Since this report was written another case of localized chronic ulcerative ileitis has been seen and operated at this hospital. The patient, age twenty-eight, entered the hospital on June 9, 1933, complaining of a tender mass in the lower right quadrant which he had accidentally palpated five days before entrance to the hospital. Two and a half weeks before entrance he had noticed a dull ache which occasionally became a sharp pain in this same region. The pain was rhythmical in character, occurring at intervals of a half minute and lasting for from three to ten seconds. The patient was conscious of this pain for approximately two weeks following its first appearance. It continued during this period with about the same degree of severity. For four days before hospitalization the pain had become much less severe. At no time was there any associated nausea or vomiting, but on careful questioning the patient stated that for the past four years there was a tendency to diarrhoea in the morning. No blood had ever been seen in the stools.

On physical examination a mass about the size of a lemon could be palpated in the lower right quadrant. The mass was tender and freely moveable. Otherwise the physical examination was negative.

Fluoroscopy and X-ray plates revealed a definite filling defect at the ileocaecal junction which was not obstructive but was definitely tender to palpation.

On the basis of physical examination and X-ray findings a tentative diagnosis of neoplasm or granuloma of the caecum was made. Because of the tenderness demonstrable both at physical and fluoroscopic examination the latter diagnosis was thought to be the more probable.

On entrance to the hospital the white blood cells were 10,000; red blood cells 4,900,000; haemoglobin 85 per cent. (Sahli); pulse 100; temperature 98.6; urine negative.

On June 10, 1933, a laparotomy was done by Doctor Phemister through a right rectus incision and a hard mass found at the ileocaecal junction. Several large, indurated lymph nodes in the adjoining mesentery could be palpated, in a line extending medially and upward. The terminal ten centimetres of ileum and proximal fifteen centimetres of caecum and ascending colon were resected and a side-to-side anastomosis made.

A frozen section at the time of operation was diagnosed as being suspicious of lymphosarcoma.

The patient made an uneventful recovery and was discharged two weeks following the operation.

The bowel resected at operation showed the following gross pathology: On opening into the lumen of the ileum the wall was found to be markedly thickened and a redundant portion of the mucosa extended for a distance of about one centimetre through the ileocaecal valve. A superficial longitudinal erosion of the mucosa of the ileum two and a half by one centimetre was found which ended sharply at the caecum. The appendix was abnormally long and curved on its mesentery in a semi-circle but was otherwise grossly negative. A mass of enlarged lymph glands was present on the posterior medial aspect of the ileocaecal junction. The largest of these nodes measured two and a half centimetres across and the smallest eight millimetres. On cross section these nodes appeared homogeneously grayish white. The caecum and colon were grossly normal.

Microscopically the picture is one of non-specific inflammation. The mucosal ulcer is shallow and has sharp margins. The mucosa adjacent to the ulcer and proximal to it for a distance of approximately ten centimetres is hyperaemic and infiltrated with polymorphonuclear neutrophils. A markedly hyperplastic Peyer's patch appears at one margin. The wall of the ileum beneath and beyond the ulcer is two to three times its normal thickness and exhibits fibrosis with a marked diffuse and focal infiltration

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consisting largely of round cells and polymorphonuclear neutrophiles in almost equal numbers. At a distance of six centimetres from the mucosal ulcer the serosa is moderately infiltrated in the same manner. This polymorphonuclear infiltration may also be seen at the tip of the appendix but does not extend through the muscularis or serosa. The cæcum and ascending colon are microscopically essentially normal. There is no evidence of tuberculosis or neoplasm in any of the sections examined.

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ANTERIOR HEMIPYLORECTOMY FOR ABERRANT PANCREATIC TISSUE OF THE DUODENUM—DIAGNOSTIC DIFFICULTIES

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Historical.—Aberrant or accessory pancreas, although recognized for a long period of time and first reported by Klob in 1859, is still quite a rare condition which is usually unsuspected until found on the autopsy table or occasionally on the operating table. Aberrant nodules which have been removed at operation have rarely been considered the etiological factors in the conditions which have made operation necessary. This is shown in the cases of Farr¹⁰ and Cafritz.⁴ Farr operated for pre-pyloric ulcer and in addition found an unsuspected aberrant nodule on the jejunum. This was excised and the defect was used as the gastroenterostomy stoma. Cafritz reported a case resembling both cholecystitis and ulcer in which laparotomy was done with a diagnosis of chronic appendicitis and an aberrant nodule was found on the anterior surface of the duodenum. Thirty-two cases of annular pancreas have been reported and there are records of not more than sixty cases of aberrant nodules of pancreatic tissue. These figures include cases where aberrant pancreas was discovered at autopsy.

Embryology.—In order to understand the origin of aberrant pancreas and to see why it is found only in certain situations, it is necessary to briefly outline the embryological development of the pancreas. There are originally three anlagen, the dorsal and the right and left ventral. The dorsal pancreas arises as a diverticulum from the dorsal wall of the foregut, slightly above the level of the common bile-duct, whereas the ventral diverticula grow down from the junction of the common bile-duct with the intestinal tube. The ventral anlagen fuse and grow to the right to meet the dorsal cell mass. If the left ventral lobe continues to grow on around the enteric tube, an annular pancreas is formed. Normally the three anlagen fuse to form one cell mass but later we shall see how aberrant nodules may develop.

Theories of Origin.—The origin of accessory or aberrant pancreatic tissue has been variously explained by different investigators. Glinski assumes a non-fusion of the three primary anlagen while Zenker postulates the presence of accessory anlagen. Thyng supports the views of Zenker and is able to demonstrate accessory pancreatic anlagen in two pig embryos. Bentler explains the condition as an atavistic phenomenon but evidence for this view is slight. Lubarsch assumes the presence of inflammatory adhesions, so that as growth proceeds, small masses of pancreatic cells are pulled away from the original cell mass. Horgan¹⁵ holds practically the same view except that he feels that the adhesions are non-inflammatory in origin. The most gen-

erally accepted explanation is that of Warthin, indorsed by Opie. They feel that buds of pancreatic tissue from the main ducts are snared off by the mesoderm of the gut and are carried away from their original site by the longitudinal growth of the intestine. This explanation seems most plausible for, as Simpson states, pancreatic tissue is never found elsewhere than along the derivatives of the foregut.

Location.—There are, in general, two types of aberrant pancreas—annular pancreas situated normally but encircling the duodenum and small nodules of pancreatic tissue, varying from several mm. to 6 cm. or more in diameter, found on the wall of the stomach, pylorus, duodenum or jejunum. Nodules have also been found on intestinal diverticula, the hepatic duct, gall-bladder or omentum and in the region of the umbilicus. Nodules are most frequently found on the jejunum, and in about one-third of the cases the nodules have been found on the wall of the stomach.

Anatomy.—Aberrant pancreatic tissue is usually found between the serosa and mucosa, the cells usually being interspersed among the muscle bundles. Growth may cause formation of a polyp in the lumen or an adenoma on the surface of the viscus depending on the direction of greatest proliferation of cells. Growth may spread the muscle, the cells pushing out to the surface, forming a roughened mass which is stippled in appearance and resembles an indurated peptic ulcer except that there is no definite crater.

Histology.—Examination of a microscopical section leaves no doubt as to the diagnosis, for typical pancreatic tissue is always present. Acini are usually seen and there may be islands of Langerhans. Large and small ducts are often present and in some cases, these ducts have been demonstrated to communicate with the lumen of the gut. The cells appear quite normal and usually contain zymogen granules so that it seems logical to suppose that secretory function is present. This is also borne out by finding occasional areas of fat necrosis.

Surgical Complications and Potentialities.—The complications arising due to the presence of aberrant pancreatic tissue may be grouped into three large divisions—mechanical, inflammatory and neoplastic.

Mechanical.—The most obvious mechanical complication is duodenal stenosis due to the presence of an annular pancreas although obstruction may occur in the pylorus, duodenum, or jejunum, due to a large aberrant mass of pancreatic cells which has pushed into the lumen and has become pedunculated. These pedunculated polypoid masses also may cause intussusception and this is most frequently seen in the jejunum. Again, interfering with the normal duodenal mechanism or acting as a source of irritation may give rise to upper abdominal symptoms. Hale¹³ goes so far as to postulate the presence of pancreatic tissue in the pyloric ring in every case of congenital pyloric stenosis. Aberrant pancreatic tissue growing toward the serosa has been said to cause diverticula but it seems more probable that pancreatic rests have developed along the course of aberrant pancreatic ducts which have been interpreted as diverticula.

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Inflammatory.—Chronic interstitial pancreatitis with or without localized fat necrosis has been reported in aberrant pancreatic tissue and absence of ducts for carrying away secretions has been given as an explanation of this pathology. Acute pancreatitis has also been observed, the etiology being as obscure as that of acute pancreatitis in the main gland. Placques of aberrant tissue have been thought to be a causative factor in the production of peptic ulcer by the proteolytic action of the secretion and this is a point which deserves more consideration than is usually given it.

Neoplastic.—Carcinomatous degeneration of aberrant pancreatic tissue is reported as a final result in some cases. Bookman¹ reports a case of duodenal carcinoma which he feels undoubtedly arose in such a manner. The history was not typical of duodenal ulcer, there was a low gastric acidity and atypical pain with the presence of a definite filling defect on röntgen studies. Laparotomy showed an extensive malignancy and microscopical section showed pancreatic cells as the origin. Cabot³ reports a death from generalized carcinomatosis, the primary source being a widespread lesion in the stomach wall without ulceration of the mucosa. Microscopical section showed the origin as probably coming from a pancreatic rest in the gastric wall.

The following two cases are reported because the patients presented clinical symptoms rather characteristic of biliary-tract disease, yet removal of aberrant pancreatic tissue from the region near the pylorus by anterior hemipylorectomy has resulted in apparent cure.

CASE I.—Mrs. H. F., aged twenty-five, entered the hospital April 12, 1933. *Present Illness.*—Four years previous to admission the patient began to have sharp attacks of severe pain in the right hypochondrium and epigastrium. The pain radiated around to the back but never went up to the shoulder. Greasy foods seemed to bring on attacks but lean meats were the most provocative. The pain was colicky in type, causing the patient to toss from side to side. Some attacks were so severe as to require morphine for relief. Vomiting accompanied some attacks but did not give relief, nor was the pain relieved by food or soda. There seemed to be excessive gas formation but she was not able to belch. Mild jaundice, especially of the scleræ, accompanied the attacks which came at first at considerable intervals and lasted from two hours to two days. Attacks had recently come more frequently, appearing about once every month but with no relation to menses.

Past History.—Appendectomy and partial left oöphorectomy six years previous to admission. Tonsillectomy five years before admission. Right oöphorectomy and uterine suspension four and one-half years before admission. Right salpingectomy and ligation of left tube two years before admission. All operations at other hospitals. Periods began at seventeen and were always regular. Has had three children and during each pregnancy has been treated for diabetes mellitus, insulin being required. During intervals, no insulin has been used and there have been no dietary restrictions. Since the birth of last child, seven years prior to admission, periods have come every two weeks. There is moderate intermenstrual leucorrhœal discharge. Chronic constipation has been present for years.

Physical examination negative except for moderate tenderness over duodenal and gall-bladder area. Bilateral cervical laceration.

Laboratory.—Blood-pressure, 120/76. Blood sugar, 87.2 per cent. Urine: 1.019, acid, few leucocytes and epithelial cells, no sugar. Blood: Hæmoglobin, 80 per cent.; red blood-cells, 4,860,000; white blood-cells, 5,800. Wassermann, negative.

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Radiography.—Moderately dense gall-bladder shadow after dye ingestion, gall-bladder lying close under liver and tip reaching fourth lumbar vertebra. No visualized calculi.

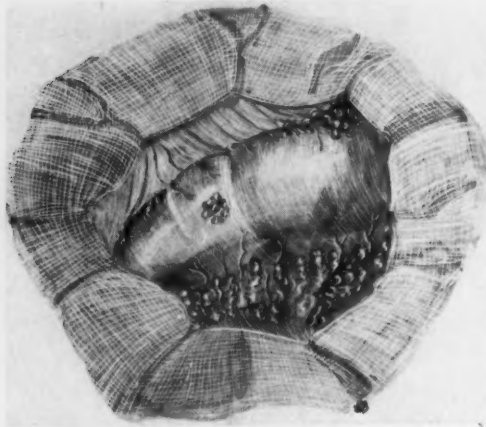


FIG. 1.—Appearance of the indurated area just on the duodenal side of the pyloric ring.



FIG. 2.—Transverse incisions just to either side of the pylorus and beyond the tumor margin are made. Dotted line indicates the connecting incision. The angle sutures have been placed.

Decrease in size by one-half following fat meal. Liver moderately enlarged. *Pre-operative Diagnosis.*—Chronic cholecystitis and cholelithiasis.

Operative Procedure.—Upper right rectus incision was made and peritoneal cavity

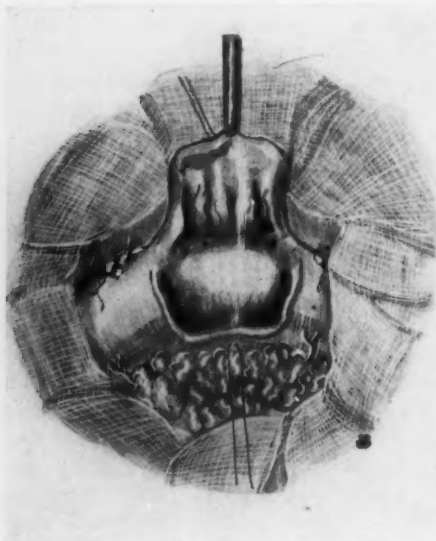


FIG. 3.—The flap of tissue consisting of pyloric ring, small amount of pyloric end of stomach and duodenum with the pancreatic tumor tissue about to be removed.



FIG. 4.—The first layer is a continuous through-and-through No. 0 chromic catgut suture. The suture comes out between the edges of the mucous membrane, thus assisting the inversion. (Mikulicz.) Over this a continuous Lembert No. 0 chromic catgut suture is placed.

opened. A hyperplastic stippled area about 1 centimetre in diameter on anterior surface of duodenum just distal to pylorus immediately presented itself in wound. Gall-bladder was thin-walled, normal in color and did not contain palpable stones. Ducts were easily

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palpable and no stones found. No adhesions but some constriction near mid-portion of gall-bladder. Diagnosis of duodenal ulcer with hyperplasia of the tissue was made and hyperplastic area was excised together with part of pyloric muscle. Anterior hemipylorotomy was done after posterior wall was shown to be free of ulcer. Wound closed in layers. The question of adenoma or pancreatic lesion was in a passing way considered.

Post-operative Care.—Nothing by mouth for forty-eight hours and then began second week Sippy diet. Pulse and temperature not remarkable and very slight pain. Post-operative course subsequently not remarkable. Fractional gastric analysis on the fourteenth post-operative day showed free 0, 0, 2, 12, 19, 32, and total 14, 10, 9, 15, 20, 41, 53. Dismissed on seventeenth post-operative day, having had no attacks of colicky pain although these should have returned with the Sippy diet of milk and cream had cholecystitis been present.

Pathological Report.—Specimen consists of mass of firm tissue 1 by 1 by 1 centimetre. No active inflammatory reaction and grossly resembles scar tissue. Microscopical section shows wall of duodenum, covered on surface by normal appearing mucosa with deep layer of Brunner's glands. Wall is distorted by number of masses of glandular tissue which lie between muscle bundles. Some of these penetrate deeply through wall and lie beneath the serosa. Numerous ducts, although opening of ducts into lumen of bowel not visualized. Glandular tissue entirely characteristic of pancreas and, in addition, few islands of Langerhans are imbedded in the mass. No inflammatory reaction or scarring.

Final Diagnosis.—Pancreatic tissue in duodenal wall. *Follow-up.*—At the end of six months, patient reported she was feeling fine with no return of symptoms.

CASE II.—Miss K. A., aged thirty-one, entered the hospital September 4, 1933. *Present Illness.*—During ten years previous to admission patient has suffered with mild but rather chronic indigestion, accompanied by belching of gas, heartburn, upper abdominal distress, but no nausea or vomiting. Patient always felt better if she avoided greasy foods and rich pastries. Food did not give relief but at times soda did. Meat seemed to bring on distress although small amounts of white meat of chicken or small amounts of fresh fish did not seem to cause any disturbance. Rough foods caused distress. She always had a tendency toward constipation but kept herself fairly well regulated with mineral oil. Always seemed to feel better if she could expel gas although she did not get complete relief from upper abdominal distress. *Past History.*—Tonsillectomy seven years ago. No other serious illnesses or operations.

Physical Examination.—Essentially negative except for moderate tenderness just to the right and above umbilicus. No muscle rigidity. Slight tenderness over appendix.

Laboratory.—Blood-pressure, 130/84. Urine: 1020, acid, no albumin or sugar, microscopic negative. Blood: Hæmoglobin, 90 per cent.; red blood-cells, 4,300,000; white blood-cells, 7,200. Wassermann, negative. Gastric analysis: Free 0, 0, 8, 16, 20, 30; total 12, 18, 26, 30, 42, 40.

Radiography.—Gall-bladder fills well, empties readily, no visible calculi. Stomach showed no abnormality. Duodenum showed tendency toward momentary spasm in region of bulb. Slight irregularity at one point which was rather constant but no ulcer niche could be made out. Colon negative.

Pre-operative Diagnosis.—Appendix dyspepsia. Cholecystitis? Duodenal ulcer?

Operative Procedure.—Rectus incision just to the right of umbilicus. A free appendix, not abnormally long or with any gross pathology, immediately presented itself. The gall-bladder was then explored, found to be normal in color. No stones in gall-bladder or ducts. Duodenum was free and on the pylorus was a thickening which was raised about 2 millimetres and measured about 8 millimetres in diameter. There was no scar and no stippling typical of ulcer. This was considered to be accessory pancreatic tissue and anterior hemipylorotomy with excision of the tumor was done. Appendix removed.

Pathological Report.—Specimen consisted of a mass of rather firm tissue measuring about .5 by .8 by .8. No active inflammatory reaction was present. Microscopical sections showed a portion of wall of duodenum with no destruction of overlying mucosa.

There were scattered areas of glandular tissue intermixed with some muscle fibre and they were characteristic of pancreatic tissue. Several islands of Langerhans were seen.

Final Diagnosis.—Pancreatic tissue in duodenal wall. *Follow-up.*—Three months after operation, patient reported that she was entirely relieved of previous symptoms and had never felt better.

Discussion.—Cases in which there is aberrant pancreatic tissue and which present symptoms are usually diagnosed as peptic ulcer, cholecystitis or malignancy, although the history and findings are rarely typical of any of these conditions. Usually the patient complains of pain in the right upper quadrant, at times severe enough to simulate gall-bladder colic, but usually mild enough to be taken for the pain of ulcer although the meal-sequence and food-relief are absent in most cases. Meat seems to be the one food which most often aggravates or brings on pain and this is explained on the grounds that protein is the greatest stimulus to pancreatic activity. Aversion to fatty or greasy foods is usually present. Poor gall-bladder function as shown by cholecystography is sometimes found and it may be argued that this also is congenital because one embryological defect is seldom found singly. Low gastric acidity has been reported in most cases and this may be of interest in relation to the peptic ulcers diagnosed in the presence of low or absent acid. Biopsy might possibly show these ulcers to have pancreatic cells at their base. Of the two cases reported in this paper, the first showed acids ranging in the upper limits of normal and the second case showed acids well within the limits of normal. In our first case, the presence of diabetes only during pregnancy has not been explained and in no other case reviewed has diabetes been reported.

Comments and Conclusions.—(1) Report is made of two cases of aberrant pancreatic tissue on the duodenal wall in which it is hoped that clinical symptoms have been removed by excision of the nodule.

(2) Aberrant or accessory pancreas is an embryological defect but clinical symptoms appear late if at all.

(3) The condition, though not frequent, is a surgical problem and may be found to have considerable significance when it is more widely recognized and understood.

(4) Possibility of aberrant pancreatic tissue as an etiological factor in peptic ulcer or carcinomatous degeneration will bear investigation.

(5) Treatment should include complete excision of the aberrant tissue so as to prevent any chance of neoplastic inflammatory or mechanical complication. Anterior hemipylorotomy was the operation of choice in the above two cases.

(6) Aberrant pancreatic tissue is usually diagnosed clinically as cholecystitis, peptic ulcer or malignancy and in these conditions, aberrant pancreas should be considered as one of the differential diagnostic possibilities.

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THE CAROTID SINUS AS AN ETIOLOGICAL FACTOR IN SUDDEN ANÆSTHETIC DEATH*

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PROBABLY most surgeons have seen the tragic accident of a death on the operating table, under N_2O and O_2 anæsthesia. I append the history of a typical case.

A middle-aged white woman presented a moderately advanced carcinoma of the breast. Careful pre-operative study revealed no pathological conditions other than the presenting lesion. The past history was good, the patient was of a phlegmatic disposition and not alarmed by the prospect of an operation. All her organs and systems functioned normally, as shown by physical and laboratory study; the blood-pressure, chemistry, and urinalysis were all normal, the accessible arteries soft, the heart and lungs normal. The usual pre-operative dose of morphine and atropine was given. The operation was a radical removal of the breast, with dissection of the axilla. In order, as I thought, to give the patient the benefit of the greatest safety, nitrous oxide was chosen as the anæsthetic. Induction was easy, without struggling, and a satisfactory level of anæsthesia was easily maintained. The patient's color, pulse, and respiration were good throughout. The blood-pressure was not noted while on the operating table.

The operation progressed without incident for about forty-five minutes. The tedious dissection of the axilla had been accomplished, the general condition remaining satisfactory, and the mass of tissue was almost removed, when, with no warning, respiration abruptly stopped. There was no mucus in the throat, no gasping, no heaving of the chest in respiratory efforts rendered futile by some obstruction to the air passages. Nor did the respirations slowly diminish until they died out. The rhythm continued regular in rate and amplitude till the last breath; moreover, the color remained good. The heart continued to beat, at first quite forcefully, then gradually faded out till it, too, stopped about an hour and a half after the breathing. During all this time artificial respiration was continued, and every means which might conceivably stimulate respiration was tried. Dilatation of the sphincter, traction of the tongue, application of cold to the chest, inhalations of CO_2 were ineffectual, and a long list of stimulant drugs were injected—all to absolutely no avail. It was impossible to obtain any response from any measure that was tried, and the effort was abandoned only when the heart ultimately failed.

This is not an isolated instance, for I have knowledge of six similar fatalities, all unpublished, which have occurred in recent years. Doubtless there are others, for the tendency is rather to hush them up. The operations included four appendectomies, one for subacute appendicitis, one herniorrhaphy, and one breast amputation.

The circumstances surrounding the deaths of all the patients I know of at first hand were very similar. In all of the cases the patients had been well studied, and all their systems were functioning normally, as shown by

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† The experiments herein reported were performed in the Laboratory of Pharmacology, University of Pennsylvania, through the courtesy of Prof. A. U. Richards. They were suggested by Prof. C. F. Schmidt, and conducted with his constant advice and criticism.

physical examination and laboratory studies. In each case the period of anæsthesia was normal, with no notable excitement or struggling, and the color was good throughout. In all cases the anæsthetic period was not unduly long, the longest period before death was about forty-five minutes—consequently the calamity occurred after the use of a comparatively moderate amount of gas. Most striking of all, there was never any warning of the approach of the fatal termination—no gasping, no change in either rate or depth of respiration, no futile efforts to breathe—merely a sudden, complete and permanent respiratory failure, with the heart continuing to beat more or less regularly in some cases for hours. Also never were any of the measures of resuscitation of any use. Caffeine, strychnine, atropine, digalen, adrenalin, lobelin, carbon dioxide and oxygen, dilatation of the sphincter, traction of the tongue—all alike were useless. Artificial respiration merely postponed the inevitable. Cardiac massage was not tried in any of these cases, but would not seem useful inasmuch as the heart beat continues.

What can be the cause of these tragic accidents? The possibilities that occur to me are: (1) Poor general condition. (2) Embolism medulla: brain—lungs. (3) Hæmorrhage, in the medulla or brain. (4) Improper administration of the anæsthetic. (5) Impurities in the gases used. (6) Asphyxia. (7) Some toxic action of N_2O , which paralyzes the respiratory centre.

I will take them up in turn.

(1) Poor general condition cannot apply. In all these cases the patients were in good general condition as far as could be determined by careful pre-operative study of the organism as a whole.

(2) Cerebral embolism seems a plausible reason for sudden death, especially in the breast-amputation case, where very numerous blood-vessels are cut and tied. Yet for an embolus to pass from the operative area to the medulla, it must traverse the lesser circulation, where the capillaries of the lungs filter out all except the most minute of microscopical emboli—hardly larger, in fact, than a red blood cell. It seems incredible that a mass small enough to traverse the lungs should yet be large enough to cause a fatal embolism in the brain. Respiration does not cease abruptly in pulmonary embolism; even in the comparatively sudden deaths, there are at least several minutes of great distress, with respiratory embarrassment, with the heart failing at about the same time as the respiration. This also requires a very large clot to close the pulmonary artery completely—larger than can come from the operative area—and there seems insufficient time for a thrombus to be built up in the pulmonary circulation. In the only case autopsied, no embolus was present.

(3) Cerebral hæmorrhage hardly seems to be a possibility, although only one of this group was autopsied. Yet the patients were all in good condition beforehand, all were comparatively young, with soft elastic arteries and normal blood-pressures. One case, whose death was even more sudden than usual, at autopsy showed no gross pathology in any part of his

brain. No sections were made; but it seems impossible that a lesion, invisible grossly, should yet exist and be so quickly fatal.

(4) Improper administration of the anæsthetic is a possible cause of death in some patients, but I think does not apply to the cases I have studied. The anæsthesia in all these cases was well conducted by people of good training and large experience.

(5) Impurities in the gases used. Although no chemical tests were made of the gases, I think the therapeutic test was convincing. Every in-

dividual death occurred with a different tank of gas—no two deaths with gas from one cylinder. In no case did a fatality occur either the first or the last time a tank was used. Every tank both of nitrous oxide and of oxygen was used for a satisfactory anæsthesia both before and after the accidents occurred. If impurities of the gases had caused death, there should have been other accidents from the use of those same tanks, and the accidents should have begun with the first use of the tank.

(6) Asphyxia did not cause death. In all the patients the color was good and the blood well oxygenated at the time of death, and asphyxia was not present. In one patient of this group a moderate degree of cyanosis appeared during induction—about the same depth as is so common during induction of N_2O and O_2 anæsthesia. If death had occurred from asphyxia, it should have occurred early in the induction when an anoxæmia was undoubtedly present. The symptoms of

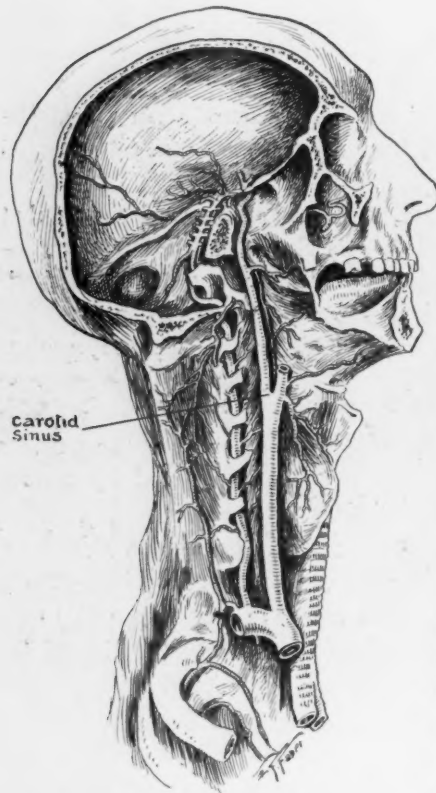


FIG. 1.—Showing location of carotid sinus, at root of internal carotid artery, behind and below angle of jaw.

asphyxia also were absent, confirming the information given by the color of the blood. In asphyxia, breathing does not cease without warning. There is a free secretion of mucus; the respirations become more and more stertorous and labored, and a tetanic rigidity sets in just before breathing stops. In other cases where the asphyxia develops less rapidly, the patient becomes pale, and the respirations grow more and more shallow until they cease.

Nothing of the kind was present in the cases I report. Anæsthesia was satisfactory, reflexes were present, the color was good and respirations were regular and full. Suddenly, with no warning, they stopped—the regular

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rhythm of breathing was interrupted and was never resumed. Nor were there any, even faint, gasps or respiratory efforts afterward. Merely inertia, and a heart which gradually failed and whose action became more and more irregular after a period of artificial respiration, until it, too, ceased.

(7) The conclusion is to me unescapable, that nitrous oxide itself has a toxic action. This toxicity has not been reported in animals, nor is it common in humans. In fact, nitrous oxide is generally considered to have no toxic action; even its anæsthetic action is supposed by many to be due wholly to asphyxia or anoxæmia.

In the hope of throwing light on this question, some animal experiments were undertaken in the Laboratory of Pharmacology at the University of

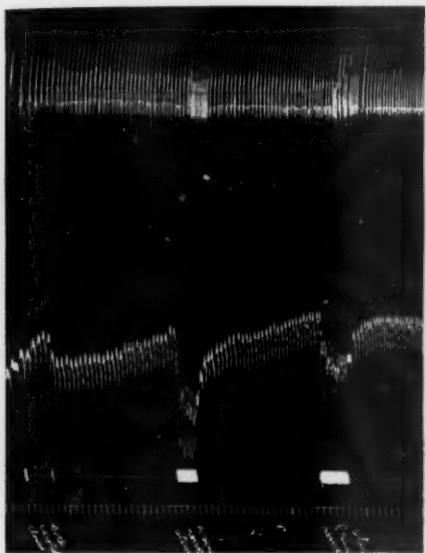


FIG. 2.—Upper tracing—pneumograph. Middle—blood-pressure in femoral artery; zero at signal line. Lowest—time marker, five seconds. Shows rise in blood-pressure and slowing respiration when endosinusal pressure is reduced to zero. Followed by fall in systemic blood-pressure and dyspnœa on electrical stimulation of each sinus nerve.

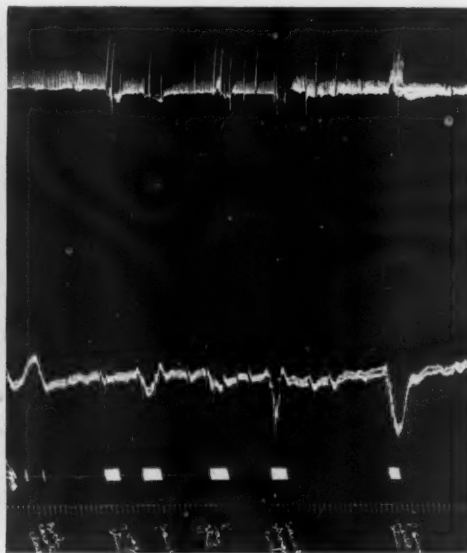


FIG. 3.—From above down—pneumograph tracing, blood-pressure in femoral artery, blood-pressure zero and signal line; time marker, five seconds. Showing contrasting effects of stimulating vagus and sinus nerves. On strong vagal stimulation, blood-pressure drops sharply, but soon escapes from the stimulus; respiration also very slow or absent. On sinus stimulation, blood-pressure does not escape, and there is marked dyspnœa.

Pennsylvania. The carotid sinus has been shown by Hering, of Cologne, to be of great importance in the normal regulation of blood-pressure and circulation; Heymans and his associates, of Ghent, and C. F. Schmidt, of the University of Pennsylvania, subsequently demonstrated also that respiration is affected by afferent impulses coming from that organ. It therefore seemed worth while to see whether the accidents under nitrous oxide might be due to a perversion of the respiratory or circulatory reflex function of the sinuses.

The carotid sinus or bulb is a dilatation of the root of the internal carotid artery (Fig. 1) which is sharply localized and is distinguished his-

tologically from the ordinary arterial wall by being thinner, and by possessing in its adventitia a rich arborization of nerves and sensory end-organs. (De Castro: *Trav. lab. de Rech. Biol. de L'Univers. de Madrid*, vol. 24, pp. 365-430, 1926. *Ibid.*, vol. 25, pp. 331-378, 1927-1928.) It is connected to the brain by its own special nerve, which is a branch of the glos-



FIG. 4.

FIG. 4.—From above down—pneumograph, blood-pressure in femoral artery, blood-pressure zero and signal line, time marker, five seconds. Another example of dyspnea on stimulation of sinus nerves, contrasted with apnea on vagal stimulation.



FIG. 5.

FIG. 5.—From above down—pneumograph, blood-pressure in femoral artery, blood-pressure zero and signal line, time marker, five seconds. Shows stimulation of stumps of cut vagus; peripheral end causes cardiac arrest, without respiratory change. Of central stump, apnea, with very slight circulatory change.

sopharyngeal. The sinus is in a state of constant tonus (Bronk and Stella: *Proc. Soc. Exp. Biol. and Med.*, vol. 29, pp. 443-445, January, 1932; Bronk, *Ibid.*, vol. 28, p. 1014, June, 1931) which increases or diminishes with varying arterial pressure within it, or which can be varied by a variety of other stimuli, especially by pressure on its wall, or by electrical stimulation of its nerve. (Fig. 2.)

It is also known that the function of the sinus can be augmented or

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diminished by the action of various drugs. Morphine, for example, increases the reflex response on stimulation of the sinus or its nerve; and the well-known bradycardia following the administration of morphine and digitalis fails to occur if the sinus nerves are cut. (Koch.) Few studies have been made of the effect of the inhalation anæsthetics; yet either has been found to abolish the reflex (Florey and Marvin); while it is increased under chloroform. When chloroform narcosis is pushed to dangerous levels, the sinus reflex persists longer than the corneal.

In fact, the familiar though misnamed "vagal pressure" of the cardiologists is actually pressure on the carotid sinus. (Figs. 3, 4 and 5.) Stimulation of the sinus or its nerve causes a fall of blood-pressure with bradycardia, with increase in the rate and depth of respiration. (Fig. 6.)

No experiments have been reported dealing with the effect of nitrous oxide on these reflexes. Consequently, it seemed of scientific interest, and possibly of practical importance to examine the matter.

In a series of nineteen dogs, under anæsthesia by N_2O and O_2 , the carotid sinuses were stimulated in various ways. Five dogs for one reason or another were unsuitable subjects and are not included. In seven of the remaining animals stimulation of the sinuses or of the sinus nerves produced a sudden transitory fall in blood-pressure, as was expected, but this was accompanied by a cessation of respiration, which in one case lasted thirty-nine minutes.

The gas used was nitrous oxide of USP quality, especially prepared for anæsthesia, and was administered with oxygen by Gwathmey's apparatus. The proportion used was as nearly as possible 20 per cent. nitrous oxide, 80 per cent. oxygen. In some experiments, a face mask was used; in other cases the gas mixture was delivered directly into a tracheal cannula, properly protected by valves. In most cases, rebreathing was not made use of; but in one experiment a glass tube of a capacity of fifty cubic centimetres was attached to the tracheal cannula. This brought the respiratory dead space



FIG. 6.—From above down—pneumograph, blood-pressure in femoral artery, blood-pressure zero and signal line, time marker, five seconds. Dog under anæsthesia— N_2O , 80 per cent., O_2 20 per cent. Several stimulations of sinus nerve give no effect. Then stimulation causes profound fall in blood-pressure, and apnoea. Artificial respiration, with recovery. Cutting the sinus nerve did not change the reaction.

approximately to what it is in the intact animal, and compensated for the volume of the trachea and pharynx short-circuited by the cannula. In a few experiments, the dog was forced to breathe against a pressure of ten centimetres of water, thinking that by raising the intrapulmonary pressure in this way more oxygen would be absorbed, and the anaesthesia would be smoother. This failed to be the case, for the dogs were usually unable to expire against the increased pressure, and became asphyxiated.

The first experiment was the most striking of all. This dog was prepared by exposing both common carotid arteries; blood-pressure, as in all cases, was taken in the femoral artery, and a pneumograph tracing was made. Gas and oxygen were supplied through a mask provided with valves, that did not permit rebreathing. The sinuses were shown to be active before nitrous oxide was started; under anaesthesia they were again tested a few times by clamping the two carotid arteries. This, by allowing the endosinusal pressure to fall to zero, produced reflexly a great rise in blood-pressure, of about fifty millimetres mercury. The second time this was repeated, when the carotid arteries were released, the pulse became very slow and irregular, blood-pressure dropped precipitously, and respirations stopped for a period; artificial respiration resuscitated the animal.

This result was so striking, and followed so closely on releasing the pressure on the common carotids, that it was sought for several times. At each trial the same thing happened. After fifteen to twenty minutes of anaesthesia, release of carotid occlusion was instantly followed by great slowing or temporary arrest of the heart, profound fall in blood-pressure, and an apnoeic period of varying length—from one minute to as long as eight and one-half minutes. Artificial respiration with pure oxygen was employed in this last case to prevent a fatal asphyxia, for the dog made no respiratory effort. After a short period, the pulse rate and blood-pressure returned to normal or even above if asphyxia was allowed to become marked. In each instance, as soon as the gas mask was removed and the animal was breathing air or oxygen, this occlusion had no unexpected results, and release of occlusion was followed by resumption of normal blood-pressure.

The stimulus for this unexpected result seemed to be the sudden distention of the carotid sinus by blood under high pressure—a sort of hammer blow—when the arterial occlusion was released. To see whether other forms of stimulation of the sinus would be effective traction on the common carotid was tried, for Hering has shown this to be an effectual stimulant of the sinus. During twenty minutes this was tried several times; at the end of that time, traction on the right carotid artery caused apnoea lasting one minute, great slowing of the heart, and a fall in blood-pressure to about twenty-five millimetres—a fall of about eighty millimetres.

By this time the dog's condition had become bad, and the experiment was terminated. It may be said in passing that though this response was elicited many times on numerous other dogs by stimulating the sinuses or their nerves in many different ways, yet on no other occasion could it be gotten by releasing the closed carotid arteries.

This sudden respiratory failure was so unexpected and so striking that this first experiment was repeated; but repeated closure of the common carotids failed to produce apnoea. It was therefore determined to try the effect of pressure on the carotid sinuses—the so-called vagal pressure experiment of Czermak. An animal whose blood-pressure had been steady for some time at about 100 millimetres of mercury, and both sinuses were active was given Nitrous oxide and oxygen when the blood-pressure rose gradually to 122, and then to 132 on closing the carotids. Stimulation of the right sinus nerve caused a fall of pressure to 110 millimetres. After twenty-six minutes of anaesthesia, while firm pressure on the right carotid sinus for forty seconds caused great inhibition of the heart

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rate, precipitous fall of blood-pressure to sixty millimetres and to fifty millimetres two minutes later with total apnoea. This lasted for three and one-half minutes but it required three minutes longer before respiration was normal again. This long period injured the animal, and it was impossible to resume the anæsthesia; though the various sinus reflexes were all normal.

This experiment was repeated on a dog who suffered with well-marked chorea. The choreiform movements disappeared only under deep gas anæsthesia, and caused great irregularities in the tracing, particularly while the subject was fresh. The sinuses, however, were very active; closure of both common carotids caused the blood-pressure to rise from 125 to 170; pressure on the sinuses produced a fall of pressure to 100 millimetres. Any influence on respiration was not noticeable, due to the chorea. After the nitrous oxide and oxygen had been started, carotid closure raised the blood-pressure to 200 millimetres. After the gas had been flowing thirty minutes, pressure on the right

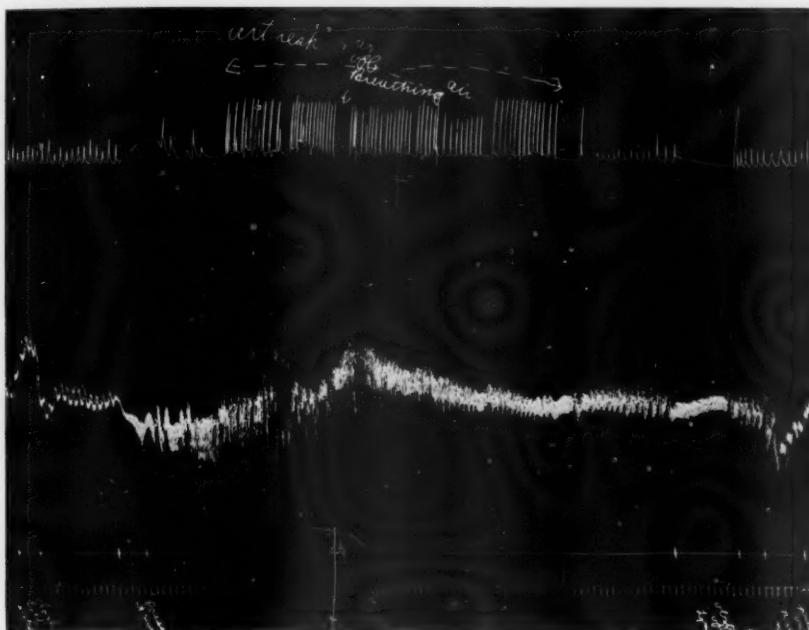


FIG. 7.—Tracings, see other figs. for explanation. Pressure on right carotid sinus causes fall in blood-pressure and bradycardia, with apnoea, lasting over five minutes. Artificial respiration continued the circulation in good condition. After recovery, pressure on larynx caused mechanical obstruction to breathing; stimulation of inferior laryngeal nerve caused slight circulatory change, no change in respiration. Dog under N_2O and O_2 anæsthesia as before.

carotid sinus, maintained for one minute and forty-five seconds, caused marked transitory fall in the blood-pressure with cardiac irregularity and slowing, and apnoea. After four minutes, one deep respiratory movement was made, and two minutes later respiration was re-established; at first very slow, deep and irregular. Finally breathing and circulation settled down to normal after a total of twelve minutes. An hour or more was now allowed for recovery, then the gas was started again, after seeing that the sinuses were still active. This time after only eight minutes of anæsthesia, pressure on the right sinus for twenty seconds produced the same circulatory collapse and apnoea lasting five minutes. (Fig. 7.) Since it had been suggested the apnoea might have been due to mechanical interference with respiration, the larynx was now strongly pulled to one side, so far as to obstruct the airway. There were no circulatory changes with this, and breathing stopped only until the airway was re-established.

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The next experiment in which positive results were obtained was most striking. In this case, after proving the activity of the preparation, nitrous oxide and oxygen were given with a mask. The blood-pressure gradually rose from 100 to 140, respirations unchanged at about twelve per minute. After fifty minutes of anæsthesia,

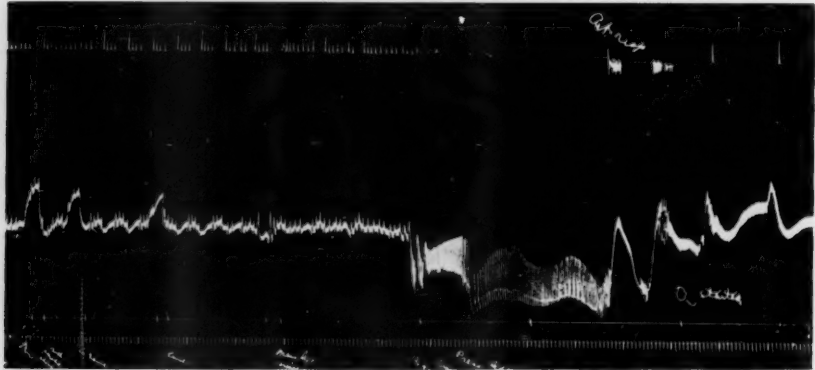


FIG. 8.—Tracings as before. Dog under N_2O and O_2 anæsthesia as before. After stimulating the sinus apparatus in various ways, at 12:01 P.M. firm pressure was made on left carotid sinus, and held for one minute, forty-five seconds. Immediate apnoea; great fall in blood-pressure and bradycardia, which soon recovered. Occasional respiratory movement, blood-pressure rising and irregular as result of asphyxia.

pressure on the right carotid sinus caused sudden circulatory collapse, with apnoea. The pressure was continued two minutes, fifteen seconds; artificial respiration was started after four minutes, and was continued with frequent interruptions. (Figs. 8, 9 and 10.) Finally, after forty minutes, breathing was resumed. During all this period, the circulation remained satisfactory, the blood-pressure holding steady at 110 millimetres until just before the end of the apnoea, when it slowly dropped to ninety.

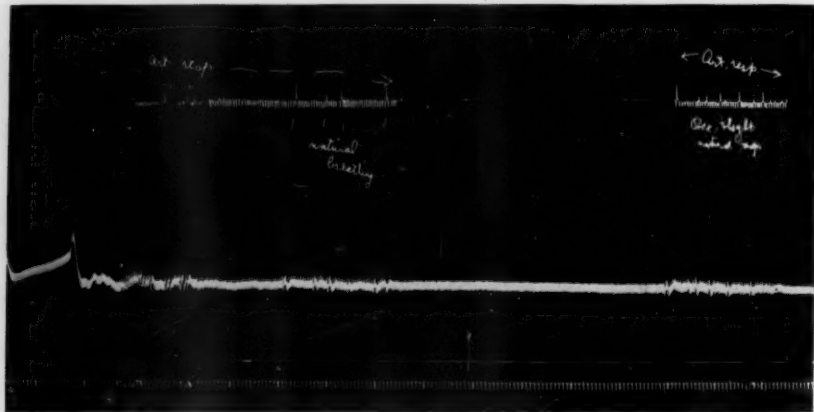


FIG. 9.—Continuation of Fig. 8. Artificial respiration used from time to time; the apnoeic interval shown is over five minutes, with no respiratory effort. However, pulse and blood-pressure remain satisfactory.

One and one-half hours were now allowed for recovery, during which the sinuses were exposed and superior laryngeal nerves were cut. Gas in the usual proportions with oxygen was started (N_2O 80 per cent., O_2 20 per cent.), but the nervous system had been injured, and breathing very soon stopped, apparently from asphyxia. This was re-established, and after a rest, the proportion of oxygen was increased to 33 per cent. The general condition remained unsatisfactory, with irregular blood-pressure.

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Finally, traction on the left carotid caused circulatory collapse with apnoea; but the heart also stopped and could not be started again. Doubtless the former prolonged apnoea had damaged the tissues.

Till now, the apnoea and circulatory collapse had resulted from stimulation of the carotid sinuses; it was now determined to stimulate the sinus nerve directly. (See Fig. 6.) After eleven minutes of anæsthesia, stimulation of the left sinus nerve with a moderate current was done for thirty seconds; a circulatory collapse resulted, and apnoea lasting two minutes. The sinus nerve was cut as soon as the stimulus was withdrawn, but this had no effect on respiration, and artificial respiration was necessary.

After allowing a period of one-half hour for recovery, stimuli were applied to the vagi, for the effect in some ways resembled vagal stimulation. Stimulation of the peripheral end of each vagus separately caused great cardiac inhibition (see Figs. 3, 4 and 5) which soon escaped from the stimulus. Stimulating the central end of each vagus caused apnoea, which lasted only until the stimulus was withdrawn.

A final experiment may be quoted, in which, after twenty minutes of anæsthesia,

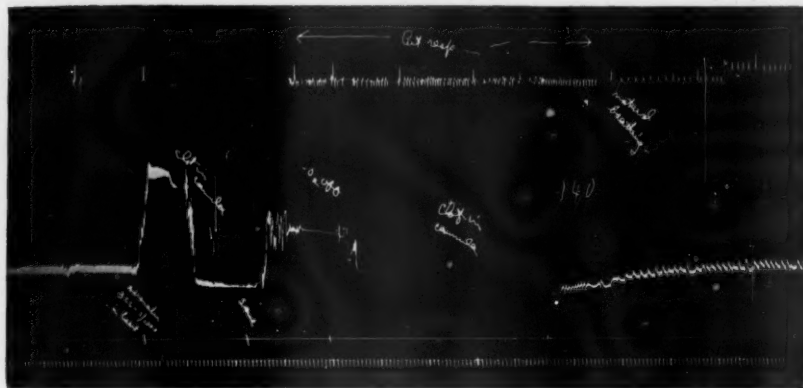


FIG. 10.—Continuation of Figs. 8 and 9. Begins with an apnoeic period of over six minutes, with only two slight respiratory attempts. Intracardiac adrenalin has no stimulating effect on respiration. Blood-pressure tracing obscured, due to formation of a clot in the cannula. When this is cleared out, blood-pressure low, but rising under the influence of artificial respiration. The total time until natural breathing resumed was thirty-nine minutes.

stimulation of the left sinus nerve produced temporary circulatory collapse, and apnoea lasting three and one-half minutes. After allowing a period of forty-five minutes for recovery, stimulation of the opposite nerve was effective; but this time the circulatory disturbance was fatal. However, the heart did not stop until some time after the respiration. The sinus nerves were cut as the circulation collapsed, but without influence on the condition.

This respiratory arrest and temporary circulatory collapse seems to us to be similar to the fatal respiratory failure that occasionally overtakes human patients in the operating room. The only apparent difference is that in dogs the respiration has been restored, while in the human this has been impossible. Doubtless the reason for this is the greater efficiency with which artificial respiration can be practised in small animals, especially forced mechanical ventilation of the lungs. Possibly a positive mechanical method such as the Drinker respirator, or something similar, would restore human patients. I have had no opportunity to try this.

The apnoea and temporary circulatory failure produced by stimulating

the carotid sinuses or their nerves occur under certain conditions only. Some of the conditions are known. It did not occur unless the anæsthesia had lasted at least 20 minutes. There seems to be some personal idiosyncrasy, for it cannot be produced in every dog. Although it is undoubtedly initiated reflexly, yet it is not wholly a reflex, for it continues after the stimulus has been removed (see Fig. 6); even cutting the sinus nerves does not shorten the period of apnoea. In some ways, it resembles the respiratory failure that occurs in anoxæmia; yet a cerebral anoxæmia produced by closing the carotid arteries is not sufficient to cause respiratory failure, unless accompanied by stimulation of the carotid sinuses, as, for example, when the sinuses are stimulated by pressure.

The exact nature of this phenomenon is still obscure. It is not clear whether it is a perverted reflex due to the direct action of N_2O on the carotid sinus or central nervous system or whether it is a result of asphyxia, or a mixture of the two. Yet it seems that certain practical conclusions can be drawn which may help to avoid this calamity.

(1) In gas anæsthesia, pressure on the carotid sinus must be scrupulously avoided. In holding the gas mask tightly to the face, the anæsthetist makes considerable pressure, and for counter-pressure, and to hold the jaw forward, often hooks his fingers on the angle of the jaw. Just behind the angle is the dangerous point; it is believed that pressure of this kind is often a precipitating factor in sudden respiratory arrest.

(2) Florey and Marvin (*Jour. Physiol.*, vol. 64, pp. 318-323, February 10, 1928) have shown that ether diminishes the sensitivity of the carotid sinus. Therefore, ether vapor added to the gases not only stimulates respiration, but also minimizes the effect of any pressure that might accidentally occur.

(3) In case of accident, all drugs are useless. The only worthwhile treatment is artificial respiration. This, if carried out mechanically in a respirator, might prolong life until normal respirations could be resumed; though opportunity has not yet occurred to try this in the human.

SUMMARY.—The occurrence of sudden respiratory failure in N_2O and O_2 anæsthesia has been described. It is shown that no yet known factor is responsible for this failure, and that hitherto no treatment has been of any avail. Animal experiments are mentioned, in which this sudden arrest of respiration was duplicated by stimulation of the carotid sinuses in various ways. This experimental respiratory failure seemed in all respects comparable to that occurring clinically.

The suggestion is made (1) that this accident can be avoided by scrupulous care to avoid pressure on or just behind the angle of the jaw; (2) that the addition of ether vapor to the gas will render the sinus less responsive to any accidental pressure; and (3) that if respiratory failure should occur, mechanical artificial respiration with a respirator is the treatment which offers the best promise of success.

THE RELATION OF POST-OPERATIVE PARALYTIC ILEUS TO MORTALITY IN ACUTE APPENDICITIS *

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THE term "paralytic ileus" is used in this paper to denote that terminal phase of small intestinal distention in which, as the result of accumulation of gas and toxic material within the lumen of the intestine and the resulting interference with blood supply, there is a complete loss of muscle tone. Clinically, paralytic ileus is characterized by prostration, extreme toxicity, failure of the intestinal musculature to respond to any form of stimulation, and early death. Paralytic ileus is preceded by varying grades of intestinal distention. Here muscle tone is still present as evidenced by attempts at contraction and response to stimuli. There is little interference with blood supply, and there is not the picture of marked toxicity. This is the stage of discomfort and is a source of danger only in that, if unrelieved, it may progress to the stage of true paralytic ileus. Post-operative distention may follow any intra-abdominal operative procedure but it is most commonly encountered in the presence of an intraperitoneal inflammatory process, especially where there is an associated peritonitis.

During the past four years a study has been carried on, on the wards of the First Surgical Division, Bellevue Hospital. The objects were: First, to determine the immediate causes of death in acute appendicitis, and second, to devise means for the avoidance of complications which might have a direct bearing on mortality. Our cases were classified into five groups:

- (1) Acute appendicitis.
- (2) Acute appendicitis with acute local peritonitis.
- (3) Acute appendicitis with peritoneal abscess.
- (4) Acute appendicitis with acute diffuse peritonitis.
- (5) Acute appendicitis with progressive fibrinopurulent peritonitis.

In going over the records it was found that the mortality in groups 1 and 2 was low, as was to be expected. The most frequent complication was post-operative distention with accompanying gas pains. In group 3, the major complication was likewise paralytic distention, seconded by acute obstruction due to adherence of a loop of intestine to the abscess wall. The mortality in this group was somewhat higher. In groups 4 and 5, acute appendicitis with diffuse peritonitis and progressive fibrinopurulent peritonitis, the mortality was found to be distressingly high. For the period 1920-1930, inclusive, our diffuse peritonitis mortality averaged 30 per cent. This represented a total

* Read before the New York Academy of Medicine, Section of Surgery, January 6, 1933.

of sixty-three cases with nineteen deaths. There were four cases of progressive fibrinopurulent peritonitis with four deaths. Thus, during this eleven-year period, there were twenty-three deaths from acute appendicitis with acute diffuse or fibrino-purulent peritonitis on the wards of the First Surgical Division. In analyzing these cases, it was found that in sixteen of the twenty-three, death was either the direct result of paralytic ileus or post-operative distention played a major rôle in the course of the disease. With these facts in mind it seemed reasonable to suppose that the elimination of paralytic ileus as a post-operative complication would result in a lowering of the mortality in these groups.

A preliminary report of this work appeared in September, 1932.* Here it was assumed that following any abdominal operation there was a period of hypotonia of the intestinal musculature of greater or less degree and duration. It was felt that if the normal tone could be maintained throughout this hypotonic period the smooth muscle of the intestinal wall would again assume its normal function and distention could be avoided. If measures were to be successful they must be in the nature of prophylaxis rather than a cure for the condition once it had arisen. Pituitrin was used in our first experiments: a routine of six doses, the first given intramuscularly directly following operation and continued at intervals of four hours for six doses. It was soon found that whereas this scheme sufficed in many cases, notably early appendicitis, the dosage was not sufficiently prolonged to prevent distention in biliary cases and cases in which there was a complicating peritonitis. Also in certain cases distention appeared before the initial dose had been given.

In a second series composed of fifty biliary cases and fifty acute appendices, with and without peritonitis, the initial dose of pituitary was given before operation where general anaesthesia was used; directly following operation where spinal anaesthesia was employed. In this series "pitressin" was substituted for surgical pituitrin as it seemed more specific for our purpose in that it contained all the elements of the old pituitrin save the oxytocic or uterine. Clinically, there had been no evidence of increased peristalsis where pituitary extract had been given in the presence of normally contracted intestine. In twelve biliary cases observations were made in the open abdomen. There was a gradual shrinking of the small intestine coming on fifteen to twenty minutes after the intramuscular administration of pitressin. This was not peristaltic in nature, and was maintained throughout the operation. It produced an extremely "quiet" abdomen and made for an easy closure of the peritoneum. In this series of 100 cases there was no instance of paralytic ileus and only moderate distention in ten cases.

During the past eighteen months there have been 112 cases of acute appendicitis, with and without peritonitis. Of this number sixty-two were classified at operation as acute appendicitis, eight as acute appendicitis with local peri-

* Potter, Philip C., and Mueller, R. Sterling: Posterior Pituitary Extract in the Prevention of Post-operative Intestinal Distention. *ANNALS OF SURGERY*, vol. 96, p. 364, September, 1932.

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tonitis, twenty-six as acute appendicitis with peritoneal abscess, fourteen as acute appendicitis with acute diffuse peritonitis, and two as acute appendicitis with progressive fibrinopurulent peritonitis. In this series pitressin was given as routine; one ampoule intramuscularly before operation where general anaesthesia was used; directly following operation with spinal anaesthesia. Groups 1 and 2 received an average of eight doses; group 3, twelve doses. In the diffuse peritonitis groups dosage was continued for considerably longer periods. In this series of 112 cases there was no instance of paralytic ileus. There were seven deaths, a mortality of 6 per cent. These deaths were confined to groups 3, 4 and 5, that is, the abscess and diffuse peritonitis groups. Among the twenty-six abscess cases there were four deaths, a mortality of 15 per cent. The first was due to a mechanical obstruction, relieved by enterostomy, but followed by massive pneumonia and death. This was confirmed at autopsy. In the second case, complicated by diabetes, there was a mesenteric thrombosis, likewise proved at autopsy. In the third case a mechanical obstruction developed at the site of the abscess. This was not relieved by ileostomy. Post-mortem revealed a double-loop obstruction, only one loop of which had been drained. The fourth death was in the case of a small boy with a large, well-walled-off abscess. Simple drainage was done. Within a few hours the temperature rose to 109° and death occurred in twelve hours. No autopsy was obtained.

Among the fourteen cases of acute diffuse peritonitis there were two deaths, a mortality of 14 per cent. In the first, a diffuse peritonitis with gangrene of appendix and adjoining caecum, a fecal fistula developed on the tenth day and there followed a slow death from sepsis. No autopsy was obtained. The second death was due to pneumonia and occurred on the fourth post-operative day.

There were two cases of fibrinopurulent peritonitis with one death. In this case jaundice appeared on the day following operation, and death occurred on the fourteenth day. Autopsy showed a diffuse fibrinopurulent peritonitis, subphrenic abscess, multiple liver abscesses and gangrene of the caecum. In no instance was death the result of paralytic ileus, nor did distention play an important rôle save where there existed a mechanical obstruction or a thrombosis.

During the past eighteen months pitressin has been employed prophylactically in 550 abdominal operations. In this series there has been no paralytic ileus. We have observed no so-called "pituitrin shock." It has been found that the presence of a post-operative pneumonitis neutralizes to a marked degree the effect of pituitary extract. In the event of this complication, pitressin is given every two hours instead of at four-hour intervals, with colon irrigations as indicated. In observing the acute diffuse peritonitis group it has been a source of surprise how little toxicity may result from the presence of large amounts of pus within the peritoneal cavity in the absence of distention. This was particularly striking in the case of fibrinopurulent peritonitis which recovered. At operation under spinal anaesthesia, a gangrenous

appendix and lower cæcum were found. There was thick colon pus throughout the right half of the abdomen and a large pelvic collection. The liver, gall-bladder and stomach were covered with plastic exudate. The intestinal coils were bright red and glued together by heavy plaques of fibrin. The appendix was removed and the lower cæcum turned in and sutured. On the tenth day, owing to a partial disruption of the wound, the abdomen was once more opened and the pathology found to be essentially the same as at the time of the first operation. In spite of the extensive involvement of the peritoneal cavity, this patient ran an unusually smooth course and was discharged on the twenty-ninth day.

Before closing, several points in technic should be emphasized. (1) Pitressin must be given intramuscularly, preferably into the deltoid. If given subcutaneously, it is often absorbed too slowly to produce the desired effect. (2) The initial dose must be given in the presence of non-distended intestine. Hence with general anæsthesia the first dose is given at the beginning of the operation. (3) The administration of pitressin must be continued at regular intervals throughout the "hypotonic period." This period varies in duration and no set rules can be laid down. However, it has been our experience that in early appendicitis without peritonitis, eight doses are sufficient. In biliary cases twelve doses are ordered. In several of our peritonitis cases we have continued the use of pitressin for two weeks or more. (4) No cathartics or enemas are given until pitressin has been discontinued. Following the final dose a colon irrigation is ordered. Finally it must be evident that the carrying out of any procedure such as the one outlined cannot be left entirely to the nursing staff. Periodic examination of the abdomen is necessary. Where early distention is noted, an additional ampoule of pitressin is ordered with a colon irrigation if necessary.

Summary.—The highest mortality in acute appendicitis is in the diffuse peritonitis groups. Here a frequent cause of death is paralytic ileus. If paralytic ileus be excluded as a post-operative complication, a decrease in mortality may be expected in these groups. A method which has proved a safe and efficient means of excluding paralytic ileus as a post-operative complication is described.

NOTE.—The series now (February, 1934) comprises 222 cases of acute appendicitis with and without peritonitis. There have been nine deaths, a mortality of 4 per cent. In the diffuse peritonitis group there have been twenty cases with three deaths, a mortality of 15 per cent.

FEMORAL HYDROCELE

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ALMOST every abdominal viscus has been reported in the sacs of femoral herniæ, as well as various extraneous objects, and Murray and Keith have identified in the femoral canal numerous diverticula of the peritoneum which contained nothing at all. One hesitates, therefore, to attempt to add anything to the literature of the subject, particularly since there can be little doubt that many surgeons have seen instances of femoral hydrocele such as are herewith reported, even though they have not, it would seem, put them on record. At any rate, an exhaustive search of the most important text-books and systems of surgery, as well as of the periodical literature through the Index Catalogue of the Surgeon General's Library and the Quarterly Cumulative Index Medicus, reveals no publication dealing with such a subject, if one is to judge from the title, except for the somewhat similar case reported by Bailey and quoted in full below. It seems justifiable, therefore, to put on the record two personal cases of femoral hydrocele from Charity Hospital in New Orleans.

CASE I.—A colored woman, aged thirty-three years, was admitted to the hospital July 22, 1929, complaining of a tumor mass in the right groin. It had appeared five years before, as a small lump, and had grown steadily until it had attained its present size; at no time had it caused pain or other symptoms. The past history was irrelevant; the patient was not married and had never been pregnant, nor did she give a story of any illness of any sort. Physical examination was essentially negative except for a mass the size of a small hen's egg located in the right femoral triangle; it was freely movable, it was not attached to the skin at any point, it was not crepitant and not pulsating, and it gave the impression of being cystic. My own tentative diagnosis was hygroma *versus* soft lipoma. The interne commented on the fact that the location, the size and the shape of the mass justified a possible diagnosis of femoral hernia, and the course of events proved his surmise correct.

Operation was done July 25, under ether anesthesia. A curved incision was made parallel and inferior to Poupart's ligament, and the mass, which lay directly below the deep fascia, proved to be a very thin-walled cyst containing translucent fluid. It could be freed of its attachments without difficulty except in the region of the femoral ring, where it extended beneath Poupart's ligament. To facilitate its removal at this point it was opened, and was seen to be lined with a glistening, translucent membrane and to contain clear, straw-colored fluid. The funnel-shaped extension of the sac lying beneath Poupart's ligament was explored with a curved clamp, and was found to lead through the femoral canal into the abdominal cavity, as was proved by the withdrawal of a portion of omentum through the canal. There was no evidence of fibrinous exudate in that portion of the sac which could be pulled through the ring, and no evidence of old or fresh inflammatory reaction. The sac was ligated high and the femoral ring was closed by the Bassini technic. Convalescence was without incident, and the patient was discharged on the thirteenth day post-operative.

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CASE II.—A colored woman, aged forty-three years, was admitted to the hospital July 31, 1931, complaining of a tumor in the left groin. Four years prior to admission a mass the size of a hen's egg had suddenly appeared in the right groin. It was associated with no symptoms, and six weeks later it had disappeared overnight, quite as suddenly as it had appeared. A year later a similar mass, though considerably smaller, had appeared in the left groin and had grown slowly but steadily ever since. The slight pain which had at first been associated had promptly disappeared and there had been no recurrence, nor had there been any other symptoms of any sort except an unexplained loss of fourteen pounds in weight within the two years preceding application for treatment. The patient was a secundipara, her younger child being twenty-nine years of age, and the menopause had occurred ten years before. Otherwise her history was entirely irrelevant.

Physical examination was essentially negative except for a soft, freely movable, fluctuant tumor, oval in shape and about the size of a hen's egg, located in the femoral triangle and apparently attached to the deeper structures only in the region of the femoral ring. A diagnosis of femoral hydrocele was made on the basis of the previous case.

Operation was done August 3, under ether anaesthesia. A slightly curved transverse incision was made across the femoral triangle below Poupart's ligament, and a cystic mass with a very thin wall, which was promptly recognized as a femoral hydrocele, was dissected free from the surrounding structures without difficulty, except, as in the first case, in the region of the femoral ring. The sac was opened, and again as in the other case was found to be lined with a glistening, translucent membrane, to contain straw-colored fluid, and to show no evidence of old or recent inflammation. Exploration through the femoral canal revealed a direct communication with the abdominal cavity. The sac was ligated high and the femoral ring was obliterated by the Bassini technic. Convalescence was uneventful and the patient was discharged on the fifteenth day post-operative.

The single case found in the literature is herewith added:

BAILEY, H.: Hydrocele of hernial sac. *Brit. J. Surg.*, vol. 15, p. 166, July, 1927:

For fourteen years L. M., aged fifty-eight, had a right femoral hernia. About a year before she came under this author's observation she developed ascites, due to cardiac failure, and the hernial sac became distended with ascitic fluid. After months of treatment with digitalis the general ascites completely abated, but the fluid in the femoral sac persisted. One week before admission to the hospital the swelling had been tapped. The distended femoral sac . . . was surmounted by a scab which had formed around the site of this puncture, and the contents had become slightly infected. Three days later the sac, which had by this time become frankly purulent, was incised. After discharging for a fortnight, the wound healed, and the patient left the hospital with no sign of the femoral hydrocele.

Comment.—Bailey's case, it will be noted, differs in several respects from the personal cases reported, in which the hydrocele was apparently primary and not secondary to some other condition. It is well known that fluid in varying amounts is likely to appear in hernial sacs in the presence of incarceration, strangulation, or certain intraperitoneal diseases, such as tuberculosis, and Seward Erdman mentions that in femoral herniæ which contain tabs of omentum fluid may accumulate and may produce a condition simulating hydrocele. Bailey's case was undoubtedly a hydrocele at the time it was aspirated, but there is every reason to believe that it had begun as an ordinary hernia and had been converted into a hydrocele when the ascites developed as the result of cardiac failure. The hydrocele, therefore, was secondary to the constitutional disease.

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The two cases from Charity Hospital are in every essential identical. While it must be admitted that negro patients of the type handled in this institution frequently give histories that are not entirely reliable, it can be taken for granted, I think, that these stories are essentially correct, and that trauma, ascites, pregnancy, previous disease, and other causes of increased intra-abdominal pressure can be eliminated as causative factors. Such inspection as was possible at operation revealed no gross evidence of pathology within the abdomen or within the sacs themselves, although unfortunately the latter point was not confirmed by histological examination. Neither mass was painful at the time of observation, and the second patient, who told a story of pain at the onset, stated that the discomfort disappeared promptly, and that at no time was it severe or disabling. In each instance the hernia was slow-growing, it was constantly present in all positions, and there were no sudden changes in size. There is no evidence in either case that the sac ever contained anything but fluid.

A rather interesting feature of the second case is the right-sided mass which was present for a few weeks a year before the left-sided tumor appeared. It is well known that peritoneal diverticula are frequently bilateral, and it is a reasonable assumption that the first tumor represented this type of hernia and that it disappeared spontaneously. It is unfortunate that the patient was vague in her recollection of its consistency.

The etiology of femoral hydrocele is impossible to establish from the facts at hand. Peritoneal diverticula are not uncommon in the femoral canal, but whether they are congenital or acquired is still to be proved. Strong evidence has been adduced on both sides of the question, but the arguments of the leading proponents of both doctrines are more notable for controversial fervor than for logic and exhibit on occasion a fine disregard for the elementary facts of physics and embryology. At any rate, the facts which have been established make it reasonable to assume that hydrocele begins as a simple hernia, a preformed diverticulum of the peritoneum, and that fluid, for some reason, accumulates and is retained within it. The fluid, rather than the hernia, needs explanation, and no explanation is entirely satisfactory.

Under ordinary conditions the fluid which is secreted by the wall of the sac and the secretion of the peritoneum which finds its way into the pouch, as in the gravity hydrocele of childhood, must flow readily back into the abdominal cavity when the recumbent position is assumed, for the neck of the sac, though narrow, is much shorter than the passage in the gravity hydrocele in the tunica vaginalis of childhood. It is necessary, then, to assume some occlusion of the neck of the sac to explain why the normal secretion of the peritoneal cavity and of the sac wall accumulated faster than it could be absorbed. In each case the opening was amply large to permit the return of the fluid to the abdomen, for it admitted without resistance the curved clamp inserted for purposes of exploration. There was no gross evidence of abdominal disease, therefore closure by an inflammatory exudate cannot be considered. Perhaps omentum or bowel occluded the opening, or

the two serous surfaces in contact with each other fused without the intervention of a fibrinous exudate, as may occur in the herniæ of childhood, the adhesions being so tenuous that they separated readily on the slight traction made when the sac was pulled down at operation. That is the most obvious explanation of the retention of the fluid within the sac, though it is far from satisfactory.

Furthermore, even assuming the occlusion of the neck of the sac, there is still to be explained the failure of balance of excretion and absorption, which is usually admirably maintained in closed serous cavities. Trauma is the most convenient explanation, and it must be granted that a tumor in the groin of an active woman is necessarily subjected to repeated minor insults, but again the explanation is inadequate. Gross evidence of hemorrhage and of inflammatory exudate, such as trauma would be certain to produce, was notably absent in both instances, and it seems highly improbable, also, that a femoral sac is ever subject to such frequent traumatism as is the tunica vaginalis in the male, in whom hydrocele of traumatic origin is a very rare condition. The etiology of femoral hydrocele, therefore, must be set down as a still unsolved problem.

THE GERMICIDAL EFFECTS OF TANNIC ACID

WITH AND WITHOUT THE ADDITION OF MERCURIAL ANTISEPTICS

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DURING recent years the tannic-acid method of treating burns has been well established. Many modifications have been made, particularly with reference to the addition of germicidal agents to the acid. This change was made because of the differences of opinions regarding the germicidal action of the acid. There is, however, a certain element of doubt as to the bactericidal action of tannic acid with and without other agents. The literature is lacking in evidence on either side of the question. The purpose of this investigation is to compare the germicidal action of different strengths of tannic acid, first without and then with the addition of mercurial antiseptics.

The bacteria used were of the common varieties, *Staphylococcus albus*, *Staphylococcus citreus*, *B. typhosus*, *B. coli*, *B. pyocyaneus*, *Streptococcus viridans*, and a short chain streptococcus. Chemically pure tannic acid of strengths 1, 2, 5, 10 and 20 per cent. in water was used. The solutions were prepared under aseptic conditions. The tannic acid itself was not sterilized because heat and chemicals which could be used in sterilization would cause changes of the acid. The culture media used throughout the experiment was prepared as follows: twenty-five grams plain agar, fifteen grams peptone, five grams sodium chloride, and three grams beef bouillon dissolved in 1,000 cubic centimetres distilled water. The whole was boiled until the constituents melted and then made neutral with sodium hydroxide, using phenolphthalein as an indicator. The agar was then sterilized twenty minutes in the autoclave. Five cubic centimetres of the agar were placed in each petri dish. All incubation was at 37.2° C. which is the optimum temperature for growth of the above organisms. In each instance where the tannic acid was used to cover the colonies, two cubic centimetres of the strength specified were used. Control plates were kept in each part of the experiment to be certain that the bacteria did not die of some other cause.

In order to determine if there was any gross chemical reaction between the mercurials and the tannic acid, metaphen 1:5,000, merthiolate 1:10,000, and bichloride of mercury 1:10,000 were placed with the different concentrations of tannic acid, 2, 5, 10 and 20 per cent. Two cubic centimetres of the mercurial were added to five cubic centimetres of the tannic-acid solutions. The merthiolate and bichloride of mercury showed no gross change when added to the tannic acid. The metaphen and tannic acid reacted with an effervescence which occurred at once in every instance, and after the solution remained for twenty-four hours the color had changed from a light brown to a dark green or brown. Due to the chemical reaction between the metaphen and the tannic acid, metaphen was not included in this work.

In the first part of the experiment the bacteria listed above were incubated for forty-eight hours. They were then checked by smear and colony formation, and in addition the colon and typhoid bacilli were checked on sugars and indol media. Two cubic centimetres of pure tannic acid of strengths indicated (Table I) were placed on the plates. The tannic acid was allowed to remain on for twenty-four hours and transplants were then made onto fresh plates which were incubated for forty-eight hours. Again

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the bacteria were checked by the method just given. The bacterial growth was luxuriant in each instance with the tannic acid of strengths 1, 2 and 5 per cent. The bacterial growth was prohibited by 10 and 20 per cent. solutions.

TABLE I

% Tannic Acid	1%	2%	5%	10%	20%	Control
<i>Staphylococcus albus</i>	Staphylococcus	Staphylococcus	Staphylococcus bacillus	—	—	+
<i>Staphylococcus citreus</i>	Spore bacillus	Staphylococcus	Spore bacillus	—	—	+
<i>B. Typhosus</i>	Short bacillus	Short bacillus	Short bacillus	—	—	+
<i>B. Coli</i>	Short bacillus	Spore bacillus Strep-bacillus	Short bacillus		—	+
<i>Streptococcus viridans</i>	Large bacillus spore bacillus	Bacillus	Streptococcus	—	—	+
Streptococcus short chain	Streptococcus short chain	Streptococcus short chain	Streptococcus short chain	—	—	+
<i>B. Pyocyaneus</i>	Bacillus	Staphylococcus	—	—	—	+

Bacteria incubated for forty-eight hours, two cubic centimeters of aqueous solution of tannic acid of strength specified placed on each dish, and then incubated for twenty-four hours. Transplants to new dishes made and read at the end of forty-eight hours' incubation.

The above procedure was repeated, using bichloride of mercury 1:10,000 instead of distilled water to make the tannic-acid solutions. There was no growth on the end plates. (Table II.)

TABLE II

% Tannic Acid	1%	2%	5%	10%	20%	Control
<i>Staphylococcus albus</i>	—	—	—	—	—	+
<i>Staphylococcus citreus</i>	—	—	—	—	—	+
<i>B. Typhosus</i>	—	—	—	—	—	+
<i>B. Coli</i>	—	—	—	—	—	+
<i>Streptococcus viridans</i>	—	—	—	—	—	+
Streptococcus short chain	—	—	—	—	—	+
<i>B. Pyocyaneus</i>	—	—	—	—	—	+

Bacteria incubated for forty-eight hours, two cubic centimeters tannic acid of strength specified in a solution of 1:10,000 bichloride of mercury placed on each dish, and then incubated twenty-four hours. Transplants to new dishes made and read at end of forty-eight hours.

The same procedure was repeated using merthiolate 1:10,000 instead of distilled water to make the tannic-acid solutions. There was no growth on the end plates. (Table III.)

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TABLE III

% Tannic Acid	1%	2%	5%	10%	20%	Control
<i>Staphylococcus albus</i>	—	—	—	—	—	+
<i>Staphylococcus citreus</i>	—	—	—	—	—	+
<i>B. Typhosus</i>	—	—	—	—	—	+
<i>B. Coli</i>	—	—	—	—	—	+
<i>Streptococcus viridans</i>	—	—	—	—	—	+
Streptococcus short chain	—	—	—	—	—	+
<i>B. Pyocyaneus</i>	—	—	—	—	—	+

Bacteria incubated for forty-eight hours, two cubic centimeters tannic acid of strength specified in a solution of 1:10,000 merthiolate placed on each dish, and then incubated for twenty-four hours. Transplants to new dishes made and read at end of forty-eight hours.

It is of interest to note that in the above work the culture media was precipitated (tanned) more by the 2 per cent. tannic acid than by the others. Precipitation occurred in the following order: 2 per cent. four plus, 1 per cent. three plus, 5 per cent. two plus, 10 and 20 per cent. one plus. The 10 and 20 per cent. changed the media to a pale brown and the 2 per cent. produced a jet-black color.

Discussion.—It is well in dealing with any substance as a therapeutic agent that the properties and mode of action of such an agent be well understood before an interpretation of the results is made. Tannic acid is an amorphous solid which is readily soluble in water and alcohol, but is practically insoluble in ether. It yields precipitates with alkaloids, gelatin, albumen and proteins,¹ and also precipitates with heavy metals. The coagulation of tissues occurs by converting the gelatines, albumen and proteins into insoluble compounds.² Solis-Cohen and Githens³ state that the bactericidal power of tannic acid varies with the ability of the cell membrane of the bacteria to resist penetration. One-half per cent. tannic acid will kill *B. coli* and *Staphylococcus aureus* in two hours but 10 per cent. will not kill anthrax in twenty-four hours.

In the first part of the experiment (Table I), unsterile tannic acid was used and the solutions were prepared under aseptic conditions. It was found that the tannic acid of strengths 1 to 5 per cent. are not sufficient germicides as evidenced by the recovery of the bacteria from the end plates in each instance. The 10 and 20 per cent. solutions were found to be germicidal within twenty-four hours. This was further borne out by other work done in the same manner.

It was also found that the tannic acid itself may be the agent of conveyance of the bacteria, as evidenced by the recovery of a number of bacteria other than the original. The 1, 2, and 5 per cent. solutions had eight contaminations, as follows: Gram-positive spore bacillus, four; Gram-positive bacillus, three;

Gram-positive streptobacillus, one; Gram-positive staphylococcus, one. The 10 and 20 per cent. solutions (Tables II and III), showed no growth on the end plates. Due to this fact, the only explanation of the large number of growths in the first part of the experiment is that the tannic acid itself carried the bacteria into the plates.

In the second and third parts of the experiment the mercurials destroyed the bacteria within twenty-four hours. (Tables II and III.) It was not deemed necessary to find how much less than twenty-four hours would be required to destroy the bacteria, because in the treatment of burns the solutions are allowed to remain in contact with the denuded areas for much longer periods.

Since there are no germicidal effects of the usual strengths of tannic acid (2 and 5 per cent.) used in treating burns, and the solutions of 10 and 20 per cent. are bactericidal, it would seem logical to use the latter. According to Seeger,⁷ the more concentrated solutions of tannic acid, even the lowest used, are highly astringent and tend to cause swelling and oedema of the tissues and too rapid fixation of the tannin. This factor alone would prohibit the use of more concentrated solutions in order to derive its beneficial bactericidal property. Therefore, the less harmful antiseptic solutions may be of some benefit, since it was found that in the less concentrated solutions of tannic acid with the antiseptics there was an inhibition of bacterial growth.

CONCLUSIONS.—(1) One, 2, and 5 per cent. solutions of tannic acid had no germicidal effect on *B. coli*, *B. pyocyaneus*, *B. typhosus*, *Staphylococcus albus*, *Staphylococcus citreus*, *Streptococcus viridans*, and a short chain streptococcus.

(2) Ten and 20 per cent. tannic acid completely destroyed the above bacteria within twenty-four hours.

(3) The tannic acid itself may be an agent for carrying the bacteria into the solutions.

(4) A greater change of culture media was caused by the 2 per cent. solution in that a more uniform change of color and precipitation of the proteins in the media was obtained.

(5) With the addition of 1:10,000 bichloride of mercury, or 1:10,000 merthiolate, to the tannic acid, all organisms were killed within twenty-four hours.

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THE TREATMENT OF VARICOSE ULCERS AND VEINS *

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VARICOSE ulcers and other late results of varicose veins of the lower extremity cause prolonged, severe pain and disability, and frequently do not receive the attention which they deserve from the medical profession. Patients with much less disabling lesions of the head, chest or abdomen receive the most careful study by the senior members of the hospital staff, while the unfortunate, who has suffered for years with a chronic ulcer of his leg, is sent to the out-patient department. Here the extensive dressing required is sometimes regarded as a nuisance, and the case is assigned to a nurse or clinical assistant to apply an ointment and supporting bandage, while the doctor spends his time considering lesions more dramatic but much less important. The patients become discouraged with the inefficiency of the treatment, and often apply bandages and local applications as best they can for themselves. The result is that chronic leg ulcers are much more numerous than is our general impression. Those who are under treatment are patients in our hospital dispensaries where they do not usually see the members of this society. They frequently have visited their local doctors, but have either failed to receive relief or have been unable to meet the expense of prolonged treatment. The long, tedious series of dressings may even make the small clinic fees an economic luxury for them. The extreme gratitude of these patients for a cure, and the way they bring in their similarly afflicted friends are a source of real satisfaction to the doctor who works with them.

The indolent leg ulcer is always a result of some underlying pathology, and it is the proper recognition and treatment of this which is requisite for success. The ulcer is merely a symptom and sign of the real disease like the glycosuria of diabetes or the cough of tuberculosis. Success in healing does not depend upon the virtues of some local application, but upon the understanding and control of the underlying circulatory difficulty. These patients suffer infinitely more and longer than many for whom we advise major operations. The doctors in the out-patient departments should carefully study each case. An ulcer which fails to heal after a reasonable time under dispensary treatment should receive the attention of the experienced surgeon, and not be allowed to become discouraged and drift away.

The ulcers to which I refer have as their basis mechanical difficulties in the venous and lymphatic circulation. I am not discussing those originating from syphilis, diabetes, *etc.*, but the underlying causes of the ulcer may be multiple. A so-called diabetic or syphilitic ulcer may be greatly benefited by the elimination of some varicose veins which are aggravating it.

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A review of the normal physiology of the venous return from the lower extremities is helpful in understanding the pathology in these cases. In the erect position, the propulsive action of the heart-beat and the arterial pressure have almost nothing to do with the venous circulation in the lower extremity. The enormous area of the capillary bed compared to that of the arterial slows the blood-current in the capillaries down to the point where there is scarcely any pressure behind it. Krogh¹ points out that a single capillary may have a larger cross-section than the tiny arteriole which supplies a large group. Also, each time an artery divides, the cross-section of its branches is greater than that of the original trunk. This results in a rapid fall of blood-pressure as we reach the terminal arteriole. The normal capillary pressure is only four to five centimetres of water at the level of the heart, a pressure insignificant when compared with the pressure required to lift the venous blood from the lower leg back to the heart when we are in the erect position. The well-known pumping action of the muscles as they press on the veins, which are equipped with valves to prevent back flow, is the propulsive force behind the venous return from the dependent extremities. The vein, with its frequent valves, is really a series of chambers, each one of which as it is compressed by the muscles empties into the one above. The normal muscular activity, being as it is alternate contraction and relaxation, is ideal for this pumping action in the veins. It is evident why we have such discomfort when we are compelled to stand still for long. Our constant shifting positions from one foot to the other, *etc.*, as we attempt it, are really reflexes to stimulate the venous circulation. Even when we deliberately dangle our legs, relaxed as much as we voluntarily can, the venous blood-pressure in the foot is far below the theoretical hydrostatic pressure which would result from its distance below the heart. The muscle tone, which is really rapid rhythmic contractions, is keeping this venous pump at work. Stand still, and the dorsal veins of the feet distend; exercise the leg muscles, and they collapse. During vigorous activity, the blood-pressure in the normal foot veins approaches zero, due to the rapid removal of blood from them by this pumping action of the muscles. Anything which cripples this venous pump, such as incompetence of the venous valves, immobilization, injury to the muscles, *etc.*, cripples the venous return from the extremity, unless the extremity is elevated approximately to the level of the heart.

The superficial veins are deprived of this muscular support. They are provided with numerous communicating veins with their valves so placed that blood can flow from the superficial to the deep veins. As soon as the venous pressure in the saphenous veins rises above that in the deep veins, blood can flow freely into the deep system where the muscular activity pumps it upward. This provides a sort of safety valve against excessive pressure in the saphenous systems as long as the valves are competent. The pressure in the normal saphenous vein is much less than the hydrostatic pressure which would result from its distance below the heart and which one would expect to be required to keep the blood flowing upward.

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Other factors in the venous circulation are the varying negative pressure in the thorax during respiration and the positive pressure in the abdomen. The effect of normal respiration on the venous return of the lower extremity while standing is insignificant, as evidenced by study of the venous pressure during respiration. Increased abdominal pressure is necessarily transmitted to the abdominal veins and the lower extremity veins as they enter the abdomen.² The extraordinary height to which this abdominal pressure can rise during violent effort has recently been measured by Murphy and Mengert.³ The abdominal veins have no valves, and this pressure can not only pump the blood on into the thorax, but also force it back against the valves in the veins of the thigh. During violent abdominal straining, our thigh muscles are also in strong contraction, which protects the deep veins but the saphenous system valves are subjected to the full pressure. The normal saphenous valves have been shown to give way at pressures of about 180 millimetres of mercury, which is not unusually high for the abdominal pressure during violent muscular work.⁸ Add to this pressure the constant strain of the hydrostatic pressure, and it is evident why varicose veins are so numerous. As the upper valves give way, more pressure is transmitted to those remaining below. As the succeeding valves give way, the whole superficial system of veins becomes wide-open channels with nothing to hinder the free regurgitation of blood downward. I do not say this mechanical back pressure is the only explanation of varicose veins but it is certainly present in every case of the common surface varices. Congenital and familial weakness of the veins certainly plays a part, and perhaps many other factors which we do not recognize and cannot control.

The source of all the difficulties resulting from simple surface varicose veins is this free downward regurgitation of venous blood towards the periphery, and the resulting capillary stagnation under pressure. The measured venous pressure in varices approaches the theoretical hydrostatic pressure which would result from the position of the vein in relation to the heart. This is still further increased by abdominal straining. This pressure is of necessity transmitted to the capillaries, as otherwise the blood current would be completely reversed in the capillaries. The blood in these veins is by analysis much higher than normal venous blood in carbon dioxide and non-protein-nitrogen content, and much lower in oxygen.⁴ The capillary permeability has been shown to be very sensitive to just these conditions, increased pressure, increase of carbon dioxide and lack of oxygen.^{5, 6} The effect of this is a profound change in the nutrition of the subcutaneous tissue. As the deep veins are intact, the disease is entirely confined to the subcutaneous tissue, which is a constant and striking clinical fact even in the most advanced stages of oedema and ulceration.

The condition of the valves in the veins communicating between the deep and superficial systems is important. When these are competent, these perforating veins are constantly removing the blood from the surface veins, and the varices, being rapidly emptied, and the pressure in them thereby kept low,

may do no more harm than greatly add to the burden of the deep veins. This is the situation when we find large varices without clinical symptoms. The extra burden on the deep circulation is undesirable, and the patient usually has fatigue and weight sensations which he does not connect with his veins, or which he has had so long that he accepts them as natural. If the communicating veins do not have competent valves, they cannot so effectively decompress the surface varices. Furthermore, blood part way up the deep veins can escape into the superficial group, where it again falls back towards the foot to reënter the deep veins, thus completing a vicious cycle.⁷ If the varices are extensive, the volume of this regurgitated blood may be more than the total volume of blood returned to the heart to receive oxygen and nutrition. This vicious cycle of old blood has been demonstrated under the fluoroscope by lipiodol injections. This situation results in nutritional changes of the entire subcutaneous tissue out of proportion to the size of visible varices.

The pathology which results from this venous back pressure on the capillary circulation is very extensive. An early and almost constant clinical sign is the common pigmentation which results from the increased capillary permeability and diapedesis of red blood-cells. I consider this pigmentation as diagnostic of impaired nutrition from varicose veins, even when they are not visible. When ulceration has occurred, the element of infection and cellulitis becomes important. These ulcers are always surrounded by a zone of acute inflammation, and frequently there are exacerbations of this infection element when large areas of the leg become very hard, swollen and painful. It is the addition to the already existing venous stasis of the element of repeated and chronic infection which results in the lymphatic blockage and elephantiasis aspect of these cases. There is a local obstruction of the lymphatics in areas of cellulitis and infection. Subcutaneous injections of India ink, which is normally rapidly removed by the lymphatic circulation, remain relatively fixed *in situ* when injected into areas of acute inflammation.⁹ This has recently been shown by Kuhns¹⁰ to be true also in joints. The absorption of substances injected into joints, which are normally quickly removed by the lymphatics, is greatly delayed if the joint is acutely inflamed before the injection.

This lymphatic obstruction within the tissues, resulting from the prolonged chronic and repeated infection in the ulcer cases, added to the existing venous difficulties, gives all the necessary elements for the gradual formation of a true elephantiasis, as described by Matas,¹¹ *i.e.*, venous and lymphatic obstruction with repeated attacks of non-pus-forming cellulitis. Halsted,¹² in studying the arm cases following breast amputations, made the same observations. Homans¹³ has always emphasized the lymphatic element in these cases. The progressive fibrosis of the subcutaneous tissue still further destroys the lymphatic circulation, and finally the whole subcutaneous tissue and skin of the leg is deprived of lymphatic as well as the normal venous drainage.

The above conditions are the late results of simple surface varicose veins,

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but there is also a group of cases where the deep veins have been damaged by a previous severe deep phlebitis or phlegmasia alba dolens. The late difficulty of the deep veins in these cases is usually not a diminution of the capacity of the veins as commonly supposed, but incompetence of the valves. These valves are just as important in maintaining the circulation of the extremities as the heart valves themselves. As soon as they are crippled, either mechanically or by infection, there is nothing to prevent the backward flow of blood in the dependent leg until the venous pressure is high enough to lift the whole column of blood up to the heart, a pressure way above the normal working pressure of the capillaries. These cases have the same difficulties as the simple surface varices, *i.e.*, œdema, ulceration, chronic infection, elephantiasis, *etc.*, but the disease is not confined to the subcutaneous tissue. Some of these cases have large surface varices, which are sometimes thought to be compensatory dilation. I believe that they are usually not compensatory but true varices which are the result of the high venous pressure. If they have a positive Trendelenburg, it is safe enough to obliterate them, but the patient receives little benefit. As a group, these cases are most discouraging to treat, as it is impossible to restore the deep venous circulation. Most of the failures of the Kondoleon¹⁷ operation for elephantiasis, which is an attempt to give the superficial tissues lymphatic and venous drainage by anastomosis with an intact deep circulation, are undoubtedly cases which fall into this class. The excision of ulcers and placing skin grafts on the deep tissues is also doomed to failure without an intact deep circulation, as well as the obliteration of the surface varices. Pressure by bandages, *etc.*, is usually of little help. Reduction of the capillary pressure and restoring the venous circulation by elevation is the only way to heal the ulcers in this group. These cases are usually easy to recognize by their characteristic history of severe œdema which involves the superficial and deep tissues dating directly from the attack of milk leg.

With these points in mind, it is much easier to develop a rational therapy for the ulcer cases. If the patient has large surface varices, and has not progressed to the stage of lymphatic block, chronic brawny œdema, and elephantiasis, remarkably rapid and permanent cure can be obtained by sclerosing the offending veins by chemical means. The ulcer in these cases usually has obvious and intimate contact with the varices. There is little or no œdema. Large veins lie just above the ulceration and very frequently under it, in which it is easy to demonstrate a positive Trendelenburg sign. The majority of the cases fall into this group. The chemical sclerosis of the offending veins by injecting them will often immediately relieve the patient of all pain and initiate healing in the ulcer. We have in our series an old lady of eighty who had had an ulcer of this type unhealed for forty years, which healed in a month after obliteration of a few veins. The larger the veins, and the more obvious their relation to the ulcer, the more satisfactory is the result of this treatment. The local application to the ulcer is immaterial.

If the ulcer patient has progressed to the point of hard, brawny œdema

or early elephantiasis, the problem is much more difficult. The varicose veins are frequently lost in the dense subcutaneous tissue. They often have no obvious relation to the ulcer, and the pathology may seem to be out of all proportion to the size of the veins. These patients are often fat, and it is frequently impossible to do a Trendelenburg test. The whole subcutaneous tissue has lost its normal venous and lymphatic drainage. Obliteration of the varicose veins in these cases should be done but it is sometimes of little benefit. If it fails, these cases with large chronic or repeatedly recurrent ulcers should be admitted to the hospital and operated upon according to the method of Homans.¹⁴ The results are very satisfactory in the twelve cases we have done. I have already shown three of them before this society. The ulcer is excised along with the underlying deep fascia and scar tissue, and Thiersch grafts are laid right on the deep tissues, whether it is periosteum, tendon or muscle. Thiersch grafts are used as the large areas involved can be covered more quickly by this method, and the results are good. The grafted area receives a new source of lymph and venous drainage from the deep circulation.^{20, 21} The hard brawny cedema disappears from this area, and the new skin, now properly nourished, is soft and normal. The principle is similar to that of the Kondoleon¹⁷ operation for elephantiasis, in which large areas of the deep fascia are excised to allow free anastomosis between the deep and subcutaneous circulation. If ulcers recur, they occur on the edge of the grafted area, or in an entirely new area, where the deep fascia is still *in situ*.

Unsuitable for this treatment is the occasional case with damaged deep veins from a severe phlebitis. It is sometimes difficult to be certain that the case falls into this group, as many of the post-phlebitis cases respond well to treatment. Extensive cedema of all the tissues, superficial and deep, is the early and ever predominant feature of these discouraging cases. DeTakats⁴ has demonstrated how the X-ray is valuable in determining if the disease is confined to the subcutaneous tissue. In a properly exposed plate, the layers of the soft parts can be made out especially when the subcutaneous tissue is very much thickened.

Trout¹⁶ published a series of cases successfully operated upon essentially according to the method of Homans, but added a point to the technic. He excised a strip of fascia above the ulcer. This combines the Kondoleon operation with Homans'¹⁹ excision of the ulcer. This involves raising flaps of skin, which in the poorly nourished diseased tissue is precarious. The only case I tried this on recently sloughed a large area of one flap.

As to technic, I believe that the skeptics of the injection treatment for varicose veins have either not tried the treatment or are unable to overcome the doctor's innate fear of intravenous medication, embolism, *etc.* Homans, long a skeptic, now acknowledges it is an excellent way to heal ulcers but believes the injected veins all recanalize. This is not true in our experience. Our series now numbers over a thousand cases, about a hundred of which I have been able to follow over three years. I admit that some veins do

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recanalize. The exact percentage is difficult to state, as it is often hard to be sure whether a vein is really the old vein or a new one. Many of the cases do, in time, form new varices. The treatment does not pretend to rid the patient of the fundamental causes for his veins, which are naturally always present, but simply to destroy the veins present at the time. We have many cases in the clinic who have been previously operated upon by distinguished surgeons who have developed large new varices, frequently right in the scars, where all the veins have been removed. Veins, on the laboratory shelf, hardened in formalin did not recanalize. This tendency to form new veins can be diminished by paying particular attention to the trunks in the thighs. I can see no necessity for ligating them, as they can be easily chemically obliterated. The internal saphenous vein may be varicose, *i.e.*, its valves be incompetent, and yet not be visibly dilated or tortuous. To make it still more difficult, it frequently cannot be palpated in the thigh fat. In these cases it can usually be found by the impulse of a fluid wave transmitted from a percussion tap on the vein below the knee, where it is dilated and tortuous. Often there are several trunks in the thigh besides the internal saphenous. If the recurrence of new veins in the leg is rapid, one should strongly suspect he has missed one or more varices in the thighs, the back pressure from which is stretching out the leg veins. The occasional recanalizing of a treated vein and the usual tendency in time to form new veins is not a serious objection to the injection treatment. It is such a simple matter to give one or two more injections as it proves necessary. A simple treatment given to an ambulatory patient is such a contrast to repeating the old operative procedure. The recurrences, taken in time, are usually much smaller than the original veins. I always explain to my patient that I am unable to eradicate the fundamental causes of his varicose veins, and am simply removing the veins which he has at the present time.

Recanalizing of injected veins can be largely prevented by good technic. It is perfectly true that the characteristic hard thrombophlebitis which follows the injection does not necessarily mean a permanent sclerosis. Too weak injection fluid, excessive dilution by blood in a large vein, too rapid circulation in the vein may cause a chemical phlebitis of insufficient severity to destroy the vein permanently.

Another limitation of the treatment is the occasional occurrence of ulcers. The solutions are all caustic. The essence of the treatment is a chemical destruction of the intima of the vein, and a drug with sufficient irritating power to do it will destroy the poorly nourished subcutaneous tissue of these cases, statements of drug manufacturers notwithstanding. I have seen these sluggish chemical burns occur after all the commonly used solutions. If the leakage has been slight and rather deep, the actual necrosis of the skin may be delayed for several weeks, the area meanwhile looking like a localized chronic infection. If the leakage has been extensive, the sloughing will be very prompt, and discouragingly slow to heal. With good technic, this accident should be rare, certainly not in over 2 per cent. of cases. I always

explain this possibility and probability to intelligent patients to protect the treatment and myself from unjust criticism. Minor points in technic diminish the possibility of slough in addition to skill in venous punctures. A very easily sliding syringe and not too small a needle help, so when the needle is properly placed, the reflux of blood into the syringe is very free and the injection can be made almost without resistance. Also, if there is the slightest doubt as to the position of the needle, or if it has slipped in and out of the vein repeatedly, one should be very quick to acknowledge failure, and either try a new vein or abandon the treatment till a later date. I have never seen serious complications from these leakage sloughs.

We have had no emboli in our series. They have been reported in the literature, and can usually be traced to cases where the injections have been given in the presence of a preëxisting phlebitis. Injections should never be given if there is any possibility of infection being already present in the veins, as the chemical irritation seems to aggravate it.

Our method of injecting veins is very simple. We have used sodium salicylate, quinine and sodium morrhuate solutions. For extensive and permanent sclerosis, I believe that sodium salicylate is the best, but the severe cramp which follows its injection frightens many patients away to clinics who are using the other two solutions, the injection of which is painless. For the ordinary varices of the lower leg, I have the patient sit on an examining table with the leg in my lap as I sit in front of him. I do the lower veins first. As the blood-current is reversed, it is much easier to obtain good sclerosis in the thighs and larger veins above, if the smaller veins below are occluded first. It is easier to identify and inject all the lower leg veins if they are treated first. I do not use tourniquets unless the veins are very large or the injection is made in the upper thigh. In large sacculations, *etc.*, where there would be excessive dilution, I attempt to inject the vein collapsed and hold the solution in the treated segment by double tourniquets. This is easily accomplished by raising the extremity and applying the tourniquets after the needle is placed in the vein. If all the veins below are blocked, good sclerosis can be obtained even in the internal saphenous in the thigh by injecting with the patient standing. I have never seen a thrombosis of the deep femoral result from an injection. The blood-current in it is so much more rapid than in the varix that the sclerosing agent is too rapidly diluted and swept on to do any harm.

For the comfort of an ambulatory patient, I recommend only one injection be given at a time and not repeated until the acute inflammation of the previous injection has started to subside. This usually means three to seven days' intervals. Under this plan the patient can usually continue with all his regular activities throughout the treatment.

If the injected vein is large, it is a distinct help to keep the vein collapsed by a firm pressure bandage or stocking during the sclerosing process. The thrombus which forms and organizes in the vein is smaller, and the hard tender swelling is much less noticeable. I have done many without compres-

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sion bandages, and it seems to me those who feel that they do not help in the large veins, have not applied them so as to keep the veins really collapsed.

The technic we have used in excision of ulcers is easy. The patient is kept in bed with the leg elevated until the œdema has been reduced as much as possible. This usually requires seven to ten days. If there is much purulent discharge and active infection, Dakin wet dressings may be used. The whole ulcer area is then excised, taking particular pains to remove all the underlying scar tissue and deep fascia. Success depends upon placing the grafts on healthy deep tissues. Any sort of graft probably could be used, but we have used Thiersch grafts as the large areas exposed can be covered rapidly this way, and the thigh from which they are taken heals rapidly without scar. The individual grafts are laid so they overlap each other and the edges of the wound, so they completely cover the raw area. They seem to take well right on periosteum, tendon and muscle. Even if take has not been 100 per cent., the region has a more normal venous and lymphatic circulation, and when it does heal, the skin will be pliable, soft and healthy. Perforated rubber tissue is placed right on the fresh graft and stuck around the edges of the wound with chloroform so it cannot shift. An even layer of gauze dressing is placed over this, and then a thick flat rubber sponge is strapped firmly over this whole area with wide adhesive tape. Further pressure is obtained by a firm circular bandage. This pressure is a very important point in getting the graft to take. The first dressing is done in five to seven days according to the odor. The patient is kept in bed with the leg elevated until the wound is healed and the grafts firm, which is usually about three weeks. When the patient first gets up, the grafts are apt to become very cyanotic, and their nutrition must be watched carefully the first few weeks in order not to lose them. Supporting pressure with sponges or absorbent cotton bandaged firmly over the area, and frequent periods of elevation help. As the new circulation establishes itself, the grafts become thicker, firmer and more like the normal skin.

To summarize, varicose ulcers, if they receive the study they warrant, can be made to heal. This can usually be accomplished by obliterating the varicose veins. In legs with chronic, brawny œdema and elephantiasis, where the lymphatic and venous circulations of the subcutaneous tissue are diseased beyond repair, permanent cures can be obtained by excising the ulcers, together with the underlying scar tissue and deep fascia, and placing skin grafts directly on the deep tissues. If the deep circulation is also diseased, this case is discouraging and elevation is the only cure.

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FRACTURES OF LOWER END OF HUMERUS *

By WILLIAM BATES, M.D.

OF PHILADELPHIA, PA.

THEORETICALLY, all fractures should be promptly reduced. Certain fractures have been allowed to wait for one reason or another before reduction was attempted, and apparently no harm done. The fractures of lower end of the humerus, especially those involving the elbow-joint, I have always considered as emergencies calling for immediate attention. This stand is taken because reduction is easier to accomplish, and becomes progressively difficult by the hour. Secondly, the earlier reduction is accomplished, the easier it is maintained. Thirdly, in the absence of injury to major vessels, the œdema associated with fractures starts to subside as soon as complete reduction occurs. This being the case, it would seem that the dreaded Volkmann's contracture could be avoided by promptness. Lastly, early restoration of functions is more readily obtained when normal alinement was accomplished promptly.

The history of the accident producing fractures of the lower third of the humerus has many times been hard to obtain. All too frequently it appears to be a "fall on the outstretched hand," and yet when we consider the classical description of various fractures, this is given as the mechanics of anything which goes wrong with any part of an upper extremity from a fracture of a carpal bone to a dislocation of the shoulder. There is, therefore, some other factor present, and because of the rapidity with which the fracture is produced, the determining factor is elusive. In the absence of direct blow from a larger object, fractures of lower third of humerus are usually not associated with fractures elsewhere in the extremity. As multiplicity of fractures is usually due to continuation of the producing force after the initial factor, the elbow is probably in partial flexion when the patient falls on the outstretched hand. The comminution of the supracondylar region is then produced by the body weight acting as a new force of injury from above in contrast to the original impact below.

Following prompt reduction under anaesthesia and under fluoroscopical visualization whenever possible, the dressing is applied with the forearm in the same relationship to the arm as when reduction was accomplished. The loss of the carrying angle in most cases is probably due to dressing the forearm against the front of the chest. The writer is indebted to Doctor Eliason for this observation and thoroughly believes that better alinement of the fracture site is maintained. The degree of flexion of forearm is likewise governed by the relationship which existed when reduction occurred.

While never having favored the very acute (Jones) flexion, if it is neces-

* Read before the Philadelphia Academy of Surgery, January 8, 1934.

sary to flex that much to get reduction then that much is necessary to maintain reduction. However, as experiences confirm the fact that with proper reduction, œdema immediately begins to subside, I have had less fear of this position. In the cases where its use was compulsory, there have been no contractures, so that my feeling is more and more in favor of rapidly preventing œdema to forestall contracture.

After the dressing is applied, the patient is held until the X-ray is developed. Believing in early reduction, I do not consider it logical to attempt reduction, dress, and wait until the next day to X-ray or see the film.

If the film is not satisfactory, the time to complete the reduction, I feel, is right then. The exceptions to this, of course, are in the types of cases where I am convinced that some form of traction is necessary to give the best result, and secondly where it is shown that open reduction is indicated.

In operating on these fractures, I have tried to convert the multiple fragments below into one group, and then consider the best way of dealing with the shaft above and the composite lower fragment. For future restoration of function, I believe the less hardware used the better the result. In one case where the capitellum was separated, I found it necessary to remove it, while in the others bone screws were used.

I have found traction necessary in very few cases but recently had a case where the usual deformity was reversed. Ordinarily, one expects to find the upper fragment anterior and the supracondylar fragments carried posteriorly. In this recent case, the supracondylar region was shattered, converting the humeral articular surface of the elbow-joint into three fragments lying anterior to the shaft of the humerus which was protruding through the skin. Open reduction in this case revealed only shell fragments which were insufficient for fixation. The wound was drained, wrist fastened to upper arm by adhesive and wide traction applied to upper forearm in line with the upper fragment. Later X-ray showed excellent alinement, but on transferring to molded splints the internal condyle was pulled slightly up and out. The condition of patient did not warrant further interference as later developments proved.

If my own experience (to which I was limited when assigned this subject) has taught me anything, it is that fractures of the lower third of humerus are so varied that few thumb rules exist for their treatment, and that each one calls for individual consideration as to the type of dressing after early reduction; and that early motion is necessary. I have found much more help in occupational therapy than in the use of baking and massage. Frequently, both aids are necessary, but when the time for active motion arrives, patients progress more rapidly under occupational-therapy supervision.

FRACTURES OF THE LEG BELOW THE LOWER THIRD *

BY ADOLPH WALKLING, M.D.

OF PHILADELPHIA, PA.

THE treatment of fractures of the lower third of the leg will ever be a subject where there are differences of opinion and it is well that this is so. Some of us are unwilling to treat all these fractures by the same method. Each fracture is a separate problem and should be studied and treated by that method which seems best to fit the individual case.

These fractures are extremely important as almost any deviation of the weight-bearing line produces disability which is sometimes sufficient to cause almost complete industrial incompetence of the individual, necessitating the use of a cane or crutches.

In no other series of fractures is accurate reduction and its maintenance so important. Any angulation of the tibial fragments will interfere with weight bearing. The weight bearing must, in order to be painless, be transmitted through smooth joint surfaces which makes essential accurate replacement of fragments involving the articular surface. It is sometimes permissible and occasionally unavoidable to have a fracture heal with some shortening. While this is undesirable the function of the leg is seldom interfered with. Perfect function is more apt to result after perfect alignment is secured early. Rotation of either fragment or lateral deviation can be prevented and must be absolutely avoided. When rigid dressings are used the foot and ankle should be kept in the position of optimum function; the foot at right angles, slightly inverted, and the arches supported.

The weakness of the muscular structure of the foot and leg makes it absolutely necessary that when weight bearing is allowed a certain amount of support be provided by elevation of the inner border of the heel or heel and sole. This support is all too often forgotten but is essential to quick return of function. Massage and movement is necessary to prevent atrophy of the muscle structure and should be insisted upon by all who treat these fractures. The period of disability is directly proportional; the earlier they are instituted the better the result.

X-rays.—Ideally, there should be an X-ray made: (1) before reduction; (2) after reduction and a permanent dressing has been applied; (3) as a follow-up. This is not always possible because occasionally an X-ray is not available beforehand because of location of the patient and for reasons of economy. Pictures should be taken as often as necessary to assure correct position of fragments during the process of healing.

Thorough skin cleansing with soap, water and alcohol and the application of a sterile dressing should be done in each fracture before a retention appara-

* Read before the Philadelphia Academy of Surgery, January 8, 1934.

tus is applied. Vesicles or abrasions may easily become infected and cause considerable anxiety as to the ultimate outcome.

Simple Undisplaced Fractures.—Often a plaster case can be applied immediately as there is never much swelling. The case should always be split along the crest of the tibia. The case need only extend to the knee. It is our rule that the case extend to the knee in fractures involving the lower third and to include the knee if the fracture is higher. If there is considerable swelling the leg is placed in a fracture box properly padded with a soft pillow and an ice bag placed over the site of fracture. The box should be opened every day and gentle massage given. The joints are moved and after the padding has been rearranged the box is closed. After the swelling has subsided sufficiently a case is applied. This is split before it hardens. The case is a light one so that one may spring it open and remove the leg for massage and movement. The massage and movement is given in the Out Patient Department by the attending surgeon. Physiotherapy is occasionally used in the aged.

In fractures of the fibula alone, weight bearing is allowed at the fourth week. Complete return of function is usual in six weeks. Weight bearing in fractures of the tibia alone is not allowed for five weeks in the transverse fractures and six to eight weeks in the oblique or spiral type. There was complete return of function in an average of nine weeks in our series. In undisplaced fractures of both bones weight bearing is begun gradually at five weeks. Full weight bearing, walking with a cane, is usually permitted at six or seven weeks. Patients returned to work with complete return of function in an average of twelve weeks. In undisplaced fractures of the maleoli an adhesive plaster boot is applied. The patient is given crutches for two weeks. Shoe correction is strictly adhered to. Good results are to be expected regardless of the method of fixation.

Fractures with Displacement.—Reduction should be accomplished immediately by the closed method under anæsthesia provided much manipulation is necessary. The anæsthetic of choice is ether. I have never used spinal anæsthesia and have used local anæsthesia on one occasion with satisfactory results when a general anæsthetic was contra-indicated. An X-ray should always be made after reduction. It is not always prudent to wait for an X-ray before reduction as in such fractures as the Pott's fracture the deformity is so typical that it is usually reduced correctly. The fluoroscope is used for visual reduction when available. It is still a matter of dispute whether every fracture should be reduced in this way. There is no question that in the complicated fractures it is essential and in nearly all fractures highly desirable, but it is a question sometimes of availability.

The preliminary X-ray is made not to tell us that a fracture is present, because the diagnosis of fracture, where there is displacement, is obvious. It tells us the exact position of the fragments and gives us a very definite idea of the mode of production and therefore what manipulations will be necessary

FRACTURES OF LEG BELOW LOWER THIRD

for reduction. If the deformity and position of fragments are obvious the preliminary X-ray is not absolutely necessary.

Failure of Reduction.—This demands either open operation or continuous traction, almost always skeletal in type. Surface traction with adhesive is next to impossible in the fracture about the ankle. Tenotomy of the Achilles tendon may be done to advantage but if skeletal traction is used it is not necessary. This should not be a complete transverse tenotomy but a partial transverse section at different levels because union takes place sooner and with more certainty.

Fixation.—We have never applied a closed case to a recent fracture immediately after reduction. Teaching a method such as this to undergraduate students may be productive of a great deal of harm as all will not be accustomed to the use of plaster. The method has no particular dangers in the hands of one experienced in plaster work. Moulded plaster splints have been employed in certain special fractures with good results; for example, transverse fracture of both bones near the epiphyseal line. This so-called Stimson splint has much to recommend it. A method of fixation that has been most useful is the fracture box. Here we have rigid splints constituting the sides of the box padded with a soft pillow. The padding can be rearranged as often as desired. Extra padding of folded towels or lint may be inserted either before or after the box is closed. Daily inspection of the fracture is facilitated by merely opening the box. The fragments are not disturbed. Massage can be given daily. The leg is elevated if necessary by elevating the box. Marked swelling is allowed to subside somewhat with the aid of ice locally and massage. A split case is applied when advisable with the foot at right angles, slightly inverted and the arch supported.

In the writer's series the average length of time before application of the case was twelve days. The patients remained in the hospital from fourteen to sixteen days. This does not include cases that were kept in the hospital for prolonged periods because of other injuries. There were fifteen cases of the so-called Pott's type during the past three years which were completely followed up. These patients returned to work on an average of fifty-seven days. One case of non-union was not included. The longest period was seventy-six days and the shortest period was forty-six days. In all cases the inner side of the sole and heel was wedged one-quarter inch to begin with. This was reduced in about three months to one-eighth inch and in six months was removed. In one case the quarter-inch wedge was so comfortable that it still remained at the end of two years. The severely comminuted fractures should be treated with skeletal traction. The fracture box here fails to come up to expectations except occasionally. It sometimes is difficult to maintain anterior posterior alignment. The fracture with separation of the posterior articular lip of the tibia may be treated in a fracture box but often this does not correct the displacement of the small tibial fragment. Mechanical fixation is sometimes necessary in this type. The fragment must be replaced at all hazards as otherwise weight bearing will be seriously interfered with.

Compound Fractures.—Most of the compound fractures of the leg occur in the lower third. If the fracture is compound from within it is only necessary to antisepticize the protruding fragment, if any, and it has been our practice to use tincture of iodine both on the leg and in the wound. A prophylactic dose of combined tetanus and gas bacillus antitoxin is given. Reduction is accomplished under anæsthesia, a sterile dressing is applied to the wound and a suitable retention apparatus applied to the leg. Immediate operative fixation of the fragments in compound fractures is rather a risky procedure. Fractures compound from without should, of course, have a thorough dé-bridement under suitable anæsthesia and a prophylactic dose of the combined serum. The number of gas bacillus infections has been greatly reduced since the prophylactic injection has been used. We have been allowing the wounds to remain open to be dakinized. The writer has not yet had the courage to use the Orr method of treatment. The Thomas splint with skeletal traction when necessary is used. Skeletal traction in any way desired is the best method of fixation, particularly if the fracture is badly comminuted or if there has been much loss of tissue. The fracture box has been used on occasion. When union has progressed sufficiently a split or a windowed case was applied.

SUMMARY.—(1) The fracture box may still be used to advantage in the treatment of fractures in the lower third of the leg.

(2) The badly comminuted type and almost all compound fractures had best be treated by skeletal traction.

(3) The articular surface of the lower end of the tibia should be returned absolutely to normal if at all possible.

(4) The normal weight-bearing line must be preserved to insure the best results.

(5) Support of the foot by wedging the inner side of the sole and heel or the heel alone is almost essential. Only too often is this neglected. If done regularly in these fractures good results are assured.

FRACTURES OF THE SHAFT OF THE HUMERUS

BY CALVIN M. SMYTH, JR., M.D.

OF PHILADELPHIA, PA.

THE material for this paper has been drawn from sixty-nine consecutive cases of fracture of the shaft of the humerus of sufficient severity to warrant hospitalization. These 69 cases occurred in a series of 191 hospitalized fractures of the humerus, constituting 36.1 per cent. of all fractures of the humerus admitted to our care over a period of three years. The sites of fracture were as follows: Upper third, 31, or 44.9 per cent.; middle third, 24, or 34.7 per cent.; lower third, 14, or 20.2 per cent. Twenty-six were transverse, 16 oblique, 7 spiral, 18 comminuted, 2 compound. No cases treated as out-patients have been considered inasmuch as cases so treated should, in the writer's opinion, be limited to those fractures without displacement of the fragments in which no reduction is required and in which the simple methods of immobilization suffice. In practically all fractures with displacement of the fragments ambulatory treatment is inadequate for the following reasons:

(1) In transverse fractures reduction is often easy but retention is often difficult or impossible by simple methods.

(2) In oblique and comminuted fractures suspension and traction or open operation, which offer the only hope of satisfactory functional and anatomical results, can be instituted only in a hospital.

TABLE I

Total fractures of humerus.....	191	
Fractures of shaft.....	69	36.1 per cent.
Site of fracture:		
Upper third.....	31	44.9
Middle third.....	24	34.7
Lower third.....	14	20.2

Methods of Treatment Employed.—Regardless of the type of fracture or the definitive treatment finally instituted, all cases are temporarily immobilized in the Thomas or Murray-Jones arm splint. This should be done by the ambulance surgeon or by the man on duty in the receiving ward before the patient is moved about in any way either for X-ray examination, undressing or any other reason. Following the X-ray examination and an accurate estimation of the situation, the method of treatment best suited to the individual case is instituted. This will depend on such factors as the type and location of fracture, whether oblique, transverse or comminuted, and whether it involves the upper, middle or lower portions of the bone.

In our experience, transverse fractures, if seen early, can usually be reduced easily, but in spite of the greatest care are extremely difficult of

retention. An illustrative case is that of a strong healthy woman of twenty-six years with a transverse fracture of the middle third. The deformity was reduced easily under the fluoroscope and perfect position obtained. She was dressed with coaptation splints, an internal right-angle splint and a shoulder cap. The arm was fastened to the side with adhesive plaster and a swathe. She was allowed to go home after the reduction and the deformity promptly recurred. Open operation was finally done.

In oblique fractures, provided there is no interposition of soft parts, satisfactory reduction can usually be accomplished by traction and suspension. The type of traction employed depends largely upon the location of the fracture. In lower third fractures, the arm is placed in abduction with the forearm in full extension in a Murray-Jones splint and skin traction applied to the arm and forearm. Six to ten pounds' extension usually suffices. In middle and upper third fractures the arm is abducted but the forearm flexed to a right angle, traction being made upon the arm alone. In our experience skin traction in fractures of the humerus will accomplish all that skeletal traction will, and a deformity which will not yield to this method, will, in most instances, indicate interposition of soft parts and require open reduction. Skeletal traction is not anything like as satisfactory in the upper as in the lower extremity where we employ it in almost every instance.

TABLE II

Type of fracture:	
Transverse.....	26
Oblique.....	16
Spiral.....	7
Comminuted.....	18
Compound.....	2

Certain comminuted fractures without great displacement of the fragments may be satisfactorily treated with plaster-of-Paris either as the circular "arm and body" case, or by molded splints, although we confess to but little success with the latter. Metal splints of the Osgood-Penhallow type are useful, but as a rule we have reserved these for later stages of the treatment.

For upper third fractures, a triangular pad in the axilla, shoulder cap, arm to the side and sling, makes a satisfactory dressing where displacement is absent or slight, and in more severe cases, the aeroplane type of splint with or without extension is often successful in bringing the lower fragment into opposition to the upper, which is almost invariably displaced outward and upward by muscle pull.

In general it may be said that deformity which can be overcome by suspension and traction will respond to these measures in about a week. If at the end of that time there is little or no improvement in position we believe that open reduction should be undertaken. Failure to respond to properly applied traction is nearly always indicative of muscle interposition and continuation of conservative measures is a waste of time to patient and surgeon alike.

FRACTURES OF SHAFT OF HUMERUS

In our series, open reduction was resorted to eleven times, or in approximately 15.9 per cent. of the cases. This is slightly higher than the open reductions upon other bones, which was necessary in 98 out of 1,000 fractures admitted to the Abington Hospital over a period of five years (1928-1932). The indications for operation in the eleven cases were as follows:

Failure to reduce by traction, 6; failure to retain after reduction, 2; compound fractures, 2; old fracture with non-union and bad position, 1. Fractures of the upper third required open reduction oftener than those involving the middle or lower thirds.

In the conduct of the operation certain general rules must be followed. Exposure must be adequate and the incision so placed that the shaft may be exposed for considerable distance without injury to the radial nerve. We have found the approach advocated by Henry to be very satisfactory. Some form of internal fixation is always necessary and our individual preference is for the Sherman steel plate, although many surgeons prefer silver wire or beef bone plates and screws. Before applying any form of internal fixation it is necessary to determine the position of the arm in which the fragments are most easily retained, or in other words, to secure the best muscle balance possible. In this respect, although certain standard positions will be applicable to most fractures, in a sense each case is a law unto itself. This has an important bearing on the dressing of the extremity after operation, for no matter how firmly fragments may be secured by plates, screws, wires or other devices, unless the part be dressed in proper muscle balance, the maintenance of good position will be difficult or impossible. This factor is of the utmost importance in the comfort of the patient after operation. It is our practice to immobilize these cases in plaster-of-Paris, the plaster including the hand, forearm and chest. Patients treated by open operation are permitted to be up in a chair in ten days and usually leave the hospital in fourteen days, returning at the end of four weeks for observation.

TABLE III
Methods of Treatment

Suspension and traction.....	28
Axillary pad and swathe.....	17
Splints (including plaster).....	13
Open operation.....	11
Total.....	69

Whether the case has been treated by suspension and traction or by open operation, union has usually progressed in four weeks to the stage where the fragments are firmly held by callus, and simple and lighter dressings may be substituted for the more cumbersome ones. It is at this time that we employ the metal splints previously referred to. Gentle massage and passive motion are now started and the dressings are changed completely once a week. More frequent revision accomplishes nothing in this type of fracture and may do

harm. All dressings may usually be dispensed with at the end of eight weeks. In patients who are careful and intelligent, a sling is often all that is required after six weeks.

In the sixty-nine cases herein considered the only case of non-union was in a patient seen for the first time twelve weeks after the original fracture. A satisfactory result was obtained after an osteoperiosteal bone graft was done. Two cases developed musculospiral paralysis but made complete recoveries. The hospitalization time averaged 18.3 days. The above were the only complications encountered and all other cases obtained excellent results.

In conclusion we wish to reiterate our belief that fractures of the shaft of the humerus should be hospitalized whenever possible; that open reduction should be done as soon as it has been demonstrated that conservative measures are not accomplishing reduction and not postponed for weeks; that the importance of immediate splinting and the early institution of definitive treatment applies here as it does in all long bone fractures.

TRANSACTIONS

OF THE

PHILADELPHIA ACADEMY OF SURGERY

STATED MEETING HELD NOVEMBER 6, 1933

The Vice-President, DR. WALTER E. LEE, in the Chair

CALVIN M. SMYTH, JR., M.D., Recorder

ACUTE STREPTOCOCCIC OSTEOMYELITIS FOLLOWING A SIMPLE FRACTURE OF THE FEMUR

DR. WALTER ESTELL LEE and DR. J. T. F. GALLAGHER reported the case of a girl of ten years, previously in good health, who was admitted to the Pennsylvania Hospital November 6, 1932, immediately following an automobile accident. There was an oblique fracture of the left femur about six inches below the greater trochanter (Fig. 1), in which the distal fragment was angulated fifteen degrees outward. There was a large hæmatoma at the site of the fracture but no break in the skin. The extremity was suspended in a Russell extension apparatus and the course was uneventful for the first three days following admission, when the temperature rose to 101° F., the pulse became rapid and the child became irrational. Examination of the head revealed a decrease in the superficial swelling and contusions of the scalp, but the left ear-drum showed a fiery injection along the handle of the malleolus. The drum did not bulge, however, and it was not incised. The right ear was not remarkable. The patient ran a hectic fever which had gradually subsided ten days after the onset with a disappearance of the signs of otitis and an apparent general improvement.

On the thirteenth day the temperature again rose sharply and there was extreme tenderness with moderate swelling and inflammation at the site of the fracture. A fluctuant mass pointed on the postero-lateral surface of the thigh which was aspirated and 320 cubic centimetres of thick grayish pus mixed with blood were withdrawn. Hæmolytic streptococci grew on the culture. Following the aspiration the swelling promptly reappeared and another tap was made, containing 350 cubic centimetres of pus and blood similar to that obtained at the first aspiration. The hectic fever continued, the fluctuant mass reappeared and two weeks after the development of the infected hæmatoma an open drainage was done at the site of fracture. Under avertin anaesthesia incisions were made along the lateral and posterior surfaces of the thigh, and a large amount of grayish-yellow pus evacuated from beneath the subfascial planes and periosteum. The periosteum was found to be stripped back from the ends of both fragments for a considerable distance. Pus also escaped from the medullary cavity and there was no evidence of bony union. Hæmorrhage from the periosteum was profuse and the vessels could not be ligated due to the extensive necrosis of the tissues. Hæmostats were left in place and the wound packed with gauze saturated with 5 per cent. solution of dichloramine-T. The patient left the operating room in profound shock and was not expected to live.

She rallied somewhat after the intravenous administration of 5 per cent. glucose in saline and during the following days she received repeated small transfusions of citrated blood. The hæmostats were removed four days post-operatively and there was no hæmorrhage, although the wounds continued to

drain pus freely. Carrell-Dakin tubes were inserted and irrigations with normal saline solution every two hours were continued for five days, when the temperature returned to normal. The wounds healed by granulation, and bony union began to take place. The patient was removed from the extension apparatus 109 days following the accident and discharged wearing a leg brace and in excellent general condition with sufficient callus to allow active motion without weight-bearing. There was perfect alignment of the shaft of the femur with no shortening of the leg. Along the lateral and anterior aspects, however, the callus was still deficient, and there was a small sequestrum with non-union of the callus at this point. (Fig. 2.)

Eight months following the injury the child resumed her normal activities without the brace. The callus increased in density but a small sinus running through the site of the fracture containing sequestra persisted. (Fig. 3.) Ten months following the accident the patient spontaneously expelled a small spicule of bone which on microscopical examination revealed the presence of Gram-positive cocci. Following this the wound healed promptly, but only last week the sinus tract reopened with slight drainage, indicating the presence of other sequestra.

It is well known that acute osteomyelitis in adolescence is essentially a blood-borne disease, localizing in the metaphyses of long bones. An important cause of such localization was determined by Lexer, when he demonstrated the loop arrangement of the capillary vessels in the metaphysis which accounts for the slowing up of the circulation and thus favoring the lodgment of bacterial emboli. In early cases of osteomyelitis Clarence Starr demonstrated that the initial lesion is always in the juxta-epiphyseal region and follows varying degrees of epiphyseal separation. In adults there is less tendency to well-defined localization of an osteomyelitis and the disease occurs much less frequently after ossification of the epiphyses has taken place. Trauma undoubtedly plays an important rôle.

The case here reported is one of acute streptococcic osteomyelitis which followed a simple fracture of the shaft of the left femur. It exemplifies the hæmatogenous origin of the disease and the determination of the site by the trauma. It is also worthy of note as a localized osteomyelitis compared with the massive infection of bone that always takes place where the intramedullary pressure is not promptly relieved. A survey of the literature indicates the rarity of such an infection although following compound fractures and amputations it is common enough.

DR. WALTER ESTELL LEE remarked that the localization of this infection to the immediate area of the fracture is in marked contrast to the massive osteomyelitis which usually develops in pyogenic suppuration of the growing long bones. We have taught for some years that this massive destruction of the bone is primarily the result of the increase in the intramedullary pressure without the cavity of the rigid bony tube, resulting from the inflammatory response, to a point greater than that of the blood-pressure within the nutrient vessels. With the cutting off of the blood supply massive death of the diaphysis rapidly occurs. Infection, though at first a localized process, rapidly involves the necrotic bone. In this case the medullary cavity was

ACUTE STREPTOCOCCIC OSTEOMYELITIS

FIG. 1.—Röntgenogram at time of accident, showing oblique fracture at junction of middle and upper thirds of the left femur.

FIG. 2.—Röntgenogram three months following accident and after surgical wounds had healed, showing unhealthy callus and bone absorption at site of fracture.

FIG. 3.—Röntgenogram eleven months following accident, showing dense callus formation with sinus tracts and sequestra at line of fracture not sufficient, however, to mar the strength of the bone.



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decompressed at the time of the fracture, and when the blood-stream infection became localized at this point, although an increased blood supply was brought to the part, it escaped from within the bony cavity through the line of fracture, and therefore, there was no abnormal increase in intramedullary pressure. No better research could have been planned than that provided by this clinical case in which, because of decompression of the medullary cavity of the bone the infection was localized. This certainly seems to bear out our belief that the amount of destruction of bone in pyogenic osteomyelitis of the growing long bones is in direct proportion to the duration of time that increased intramedullary pressure is allowed to continue, and that by very prompt decompression of the medullary cavity by drilling holes in the cortex or by cutting a trough through the cortex, the blood supply is restored to the cortex and death of bone is minimized.

DR. HENRY P. BROWN said that a young man of thirty years was admitted to Doctor Hodge's service at the Presbyterian Hospital with a fractured femur which could not be reduced satisfactorily by traction. About seven or eight days later, on open reduction, a collection of serum was found between the ends of bone from which was obtained staphylococci in pure culture. After reduction the wound was closed without drainage, no infection followed and the patient had a primary union.

IMPERFORATE ANUS

DR. I. S. RAVDIN presented a boy seven years of age who when forty-eight hours old was admitted in the service of Dr. G. P. Muller in the Hospital of the University of Pennsylvania with the diagnosis of imperforate anus. The child was born on the second of February, 1926. The labor was about nine hours long and was unattended by any difficulties. At birth he was apparently healthy; after two days the child seemed well but no bowel movements had taken place. Examination revealed that he had no anal opening. He was well developed and had no deformities except for the absence of an anal opening. The abdomen was rotund and distended, and moderately tympanitic. Peristalsis was hyperactive. There was no anal opening nor any sign of a pit or bulge in the perineal region. The child was operated upon by Dr. I. C. Ravdin on the day of admission, with "sugar-tit" anaesthesia. An incision was made in the perineum from the posterior edge of the scrotum to the tip of the coccyx. By careful dissection and keeping quite close to the curve of the coccyx and sacrum, the incision was carried up to the cul-de-sac of the peritoneum. At that time no bulging of the non-descended rectum was noticeable. By measurement they had gone nearly six centimetres. After a little more dissection a bulge during inspiration was noticeable and there was finally disclosed a blue mass bulging into the wound. (Fig. 4.) This was opened and about 250 cubic centimetres of meconium with gas were immediately evacuated. After evacuation of the bowel it was possible to bring the gut down to the edges of the wound where it was sutured with six sutures of black silk. A piece of iodoform packing was put on either side of the wound between the rectum and the perineal tissues and a rubber tube was inserted into the lumen of the bowel.

At the conclusion of the operation the child was given 50 cubic centimeters of citrated blood by intraperitoneal route.

IMPERFORATE ANUS

No inhalation anaesthesia was given at any time during the operation and the child did not seem to suffer any great amount of pain; in fact he slept during the greater part of the time.

Two days after the operation the bowels moved three times during the night. The temperature rose sharply to 105° F. but the general condition was satisfactory. It was taking mother's milk—two ounces, every three hours. On the fourth day following the operation the condition was satisfactory although the child continued to run a temperature of about 103° F. The bowels moved two or three times that day. On the sixth post-operative day the tube and the skin stitches were removed. The wound was clean. The temperature rose to 105° F. again on this day but the general condition seemed fair. The lungs remained clear. Two days following this the temperature fell to 100° F. Ten days after operation the wound was clean and healing nicely, and the temperature was normal. There was still a granulating surface but the bowels moved regularly. From the time of discharge dilatation of the anal opening was practised but the stricture became increasingly firm.

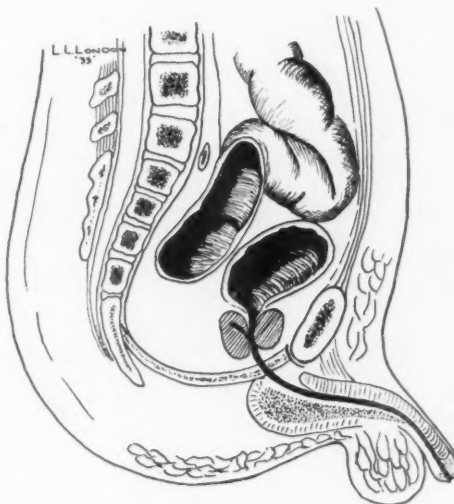


FIG. 4.—Terminal state of rectum as disclosed at operation.

On June 1, 1931, the child was again admitted to the hospital with a rectal stricture. On the following day a plastic operation was performed. The stricture on one-half of the anus was excised and the mucous membrane sutured to the skin around the anus. Fourteen days later a similar operation was done at which time was removed the remainder of the scar which was causing the stricture.

The child has grown normally and is in excellent condition. Except when his movements are loose he controls his evacuations. He attends school and participates in games with his schoolmates.

While there has been an increasing tendency to resort to inguinal colostomy for the primary operation the perineal route would seem to be the preferable exposure unless the blind end of the colon hangs free in the peritoneal cavity. If adequate exposure is obtained and the tissues are gently handled the operation should not be attended by much shock.

IMPERFORATE ANUS—END-RESULT

DR. WALTER ESTELL LEE and DR. N. P. SHUMWAY presented a male infant born in the Philadelphia Lying-In Hospital November 6, 1931. He was the first child of rather elderly parents. The delivery was prolonged and rather severe by reason of a breech presentation. Upon admission to the nursery on attempting to obtain the rectal temperature absence of the anus was discovered; this was reported immediately to the attending physician but because of the child's general condition it was determined to wait for some improvement before attempting surgical relief. After fourteen hours' observation there was some general improvement, but abdominal distention

was rapidly increasing and there was slight but persistent regurgitation of fluids; the child was restless and cried continually. Examination at the time revealed total absence of an anal dimple and there was no bulging or thrust of the perineum upon straining. Colostomy was, therefore, performed fifteen hours after delivery. Six and one-half hours later there was sufficient plastic peritoneal reaction to have securely walled off the abdominal cavity and the gut was opened longitudinally with a nasal tip cautery. There was immediate discharge of meconium. The supporting catheter was removed three days later.

The patient had no post-operative reaction and his subsequent course

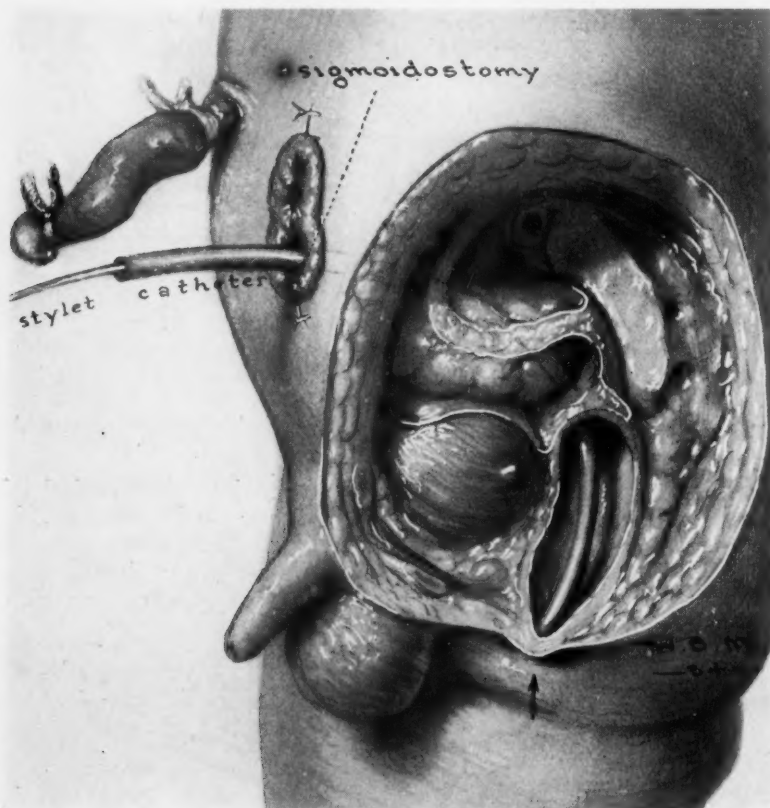


FIG. 5.—Sigmoidostomy subsequently followed by perineal section under guidance of a catheter introduced on a stylet through the opening in the sigmoid.

was most satisfactory, showing a progressive gain in weight. The only complication was a tendency toward excoriation of the skin about the wound. This was best controlled by a liberal use of vaseline-gauze dressings and the institution of an acidophilus milk formula.

On the thirty-fourth day after delivery a small catheter, No. 10F, was inserted through the colostomy opening into the distal segment of the bowel; through this, iodized oil was injected with a resulting visualization of the rectal pouch. By comparison with an opaque marker on the perineum the rectum appeared to be about one inch away; the pouch was quite clearly seen and there seemed to be a conical extension beyond the main bulb toward the anal region.

IMPERFORATE ANUS

An attempt was made to connect the rectum with the perineum on the forty-first day of life, anaesthesia being successfully obtained with whiskey water. Under fluoroscopical control a No. 16F soft rubber catheter with blunt tip was inserted into the lower loop of the sigmoid through the colostomy; some difficulty was met with in pushing the catheter into the rectum because of coiling, which tendency was obviated by threading a fine wire stylet into the tube, which also added to its opacity to the X-ray, affording very satisfactory visualization. (Fig. 5.) The exact position of the rectum having been determined by this means, incision was then made through the perineum. No muscular tissue being met with, the dissection was carried toward the rectum until the catheter could be readily palpated and finally grasped and brought out through the wound—a distance of approximately three centimetres. The rectum was not mobilized and the perineal wound was closed about the catheter with two skin sutures of dermol. By this procedure the possibility of opening into the peritoneal cavity was prevented. There was no shock post-operatively and there was a rise of temperature to only 100° on the following day; other than this there was no untoward reaction. The catheter was removed at the end of eleven days and another of the same calibre reinserted through the colostomy wound; this procedure was followed at weekly intervals thereafter for the first month after operation, when the catheter was no longer inserted from above, that portion connecting the rectum with the anus alone being maintained. On the forty-fifth day after perineoplasty there were free faecal passages by rectum. Three months after operation the child was discharged from the hospital to be followed in the Surgical Out-Patient Department; there the routine has been a weekly insertion of a mushroom catheter into the rectum.

Examination at the present date shows a well-developed and active child, weight twenty-eight pounds, with a left-side colostomy wound which occasionally discharges faecal matter. The rectal examination reveals a tract which will just admit the little finger; its wall is covered with rectal mucous membrane extending almost to the perineum. The surrounding tissue is firm and indicates that continual dilatation will be required to maintain its patency. There is, of course, no control of the evacuations since the catheter is still being inserted at weekly intervals.

Congenital imperforate anus is not a common complication of infancy, occurring but once in 5,000 to 10,000 births, however, it is a condition which being present usually demands immediate surgical treatment. Like most other congenital defects it is frequently complicated by atresia of some other portion of the intestinal tract, by cardiac malformations, or genital deformities which as a rule make the prognosis poor; even without a second deformity the outlook is not exceptionally good, for many writers upon the subject are of the opinion that the rectal anomaly itself exerts a retarding effect on the foetal development in utero. This is an important factor in the determination of the type of surgical procedure to be used, in that one is dealing with a patient who demands the least trauma possible.

The etiology of the condition lies in the embryological development of the portion of the gut at fault; this fact was most clearly elucidated by Keith in 1908, and it was upon this basis that he proposed a most satisfactory classification of the various types of deformity found in this region. Up until the tenth or twelfth week of foetal life the hind-gut and the Wolffian

body form a common cloaca; these rapidly separate and the hind-gut pouches down to meet an invagination of the proctodeum which forms the anus, fusion taking place at about the twelfth week. This process is based upon the evolutionary development of the vertebrates and really amounts to the migration of the anus from an intraclacal position, as found in the frog, to an extraclacal or perineal position. With this as a basis we are able to determine the three groups into which all deformities must fall. First: those in which the rectum empties into the urogenital tract; second, those in which the hind-gut is imperfect; third, in which the proctodeum is imperfect. It is obvious from the development of the region that any one or all three may occur with any combination of them together, and when it is further realized that the internal and external sphincters are a part of the proctodeum we must conclude that the external examination of the parts does not give any indication as to the exact condition present. Thus we may have a normally formed anus and yet the rectum be anywhere from one-sixteenth of an inch to two inches away from the perineum. This is the second fact which must influence our decision in the matter of surgical approach.

Limiting the discussion to the condition found in the case reported, namely, a combination of the second and third types, it may be said that the symptomatology of the condition varies with the time elapsing between birth and the surgical intervention resorted to; that is, if the period is lengthy the cardinal signs and symptoms of acute intestinal obstruction may be expected.

Mortality in this condition is rather high, death being certain if no operative procedure is resorted to; the figures for those operated upon give as a rule a total mortality of about 25 per cent. Brenner, reporting sixty-one cases, gave 26.2 per cent. although of these but three were of the type with complete occlusion. Of 104 cases collected from the more recent literature the total mortality was 20.2 per cent.

Of the various operations performed for the relief of imperforate anus the one which suggests itself most readily is that first employed by d'Amussat, which consists in a dissection of the perineum with search for the rectum and its mobilization so that it may be brought down to its proper position. This method is the one chiefly favored. The second route is that of colostomy with perineoplasty. A third method is that of colostomy with secondary perineoplasty. A fourth method is mentioned and condemned by all writers, that of blindly searching for the rectum with a trocar. Here the danger lies in piercing Douglas' pouch, which often lies between the perineum and the rectum, with subsequent peritonitis.

The first method described is advocated by the majority of men who have written upon the matter, and in a greater part of the statistics it has had the lowest mortality; thus in Brenner's series of twenty-nine cases operated on by direct perineoplasty the mortality was 24 per cent.; of the eleven in whom colostomy was performed the deaths amounted to 66 per cent. However, it was our feeling that in general the statistics against primary colostomy

IMPERFORATE ANUS

are deceiving in that often it had been resorted to only after perineal search had failed, and consequently the child had been subject to a great deal of trauma. With this in mind we have grouped the 104 cases from recent reports into those in whom perineorrhaphy was primary, sixty-three with a mortality of 22.2 per cent.; colostomy, twenty-two with a mortality of 18 per cent.; and the combined, sixteen with a mortality of 68.7 per cent. In this it is seen that if the last two are combined the mortality for colostomy is 39.4 per cent., whereas the corrected deaths are actually lower than the 22 per cent. found in the type of operation chiefly advocated; moreover the perineal route is successful in only 80 per cent. of the cases, the remainder requiring colostomy as a last resort. Recently, however, Wangenstein and Rice have described a method of determining the position of the rectum by X-ray which should reduce the mortality for the perineal route; they advocate the placing of the child in a head-down position in order to allow an accumulation of gas in the rectal pouch with subsequent röntgen examination to definitely localize the end of the gut. This entails a certain delay in operation, at least ten to twelve hours being required for the formation of a sufficient quantity of gas to outline the rectum; and while many believe that operation may be postponed for twenty-four to forty-eight hours, yet this idea is certainly contrary to the principles of surgery in the treatment of intestinal obstruction. Further, the intestinal tract of the child during the first thirty-six hours of life is generally thought to be sterile, and since gas to be present must be preceded by the gas-forming organisms, operation by this method is being put off until there is an infected field, a totally irrational procedure.

Primary colostomy, as performed in the case reported by us reveal no delay, was borne well by the patient, and permitted subsequent use of a method by which the rectum was accurately visualized before and during the secondary perineal repair.

The literature contains mention of similar visualization of the rectal pouch with an X-ray-opaque substance injected through the colostomy wound, by Doctor Hirschman of New York while discussing a paper by Doctor Landsman in 1926. We have not found any report of the use of an opaque catheter and fluoroscope as was done in our case at operation.

DR. I. S. RAVDIN said that his patient, of course, had no sphincter because there never had been any. The ideal thing to do for this child is the operation for anal incontinence described under several names but which should probably be credited to Harvey Stone. This operation consists in slinging two strips of fascia around the anus by passing them subcutaneously from the gluteus maximus muscles; the strips pass around the sides of the anus opposite to the muscles from which they start and interlock with each other. Control is afforded by contraction of the glutei, which in turn tightens the fascial ring. The speaker proposes to do this operation sometime in the near future.

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DR. GEORGE M. DORRANCE mentioned a patient who is at present under his care in whom the perineal operation was unsuccessfully attempted. On opening the abdomen the blind end of the bowel was found to be adherent to the bladder and colostomy, was, therefore, performed. Now there is a prolapse of about eight inches of sigmoid through the abdominal wound.

FRACTURES OF THE CAPITELLUM OF THE HUMERUS

DR. WALTER ESTELL LEE and, by invitation, DR. THOMAS SUMMEY, read a paper with the above title for which see vol. 99, page 497.

SPINAL ANALGESIA—A REPORT OF FIFTEEN HUNDRED CASES

DR. ORVILLE KING, by invitation, read a paper with the above title.

PRIMARY CARCINOMA OF THE COMMON BILE-DUCT

DR. WALTER ESTELL LEE and, by invitation, DR. H. P. TOTTEN read a paper with the above title.

TUBERCULOSIS OF THE BREAST

DR. WALTER ESTELL LEE and, by invitation, DR. HARRY G. FLOYD read a paper with the above title for which see vol. 99, page 753.

DR. I. S. RAVDIN recalled a case operated upon by the late John B. Deaver in 1920 which had been operated upon previously by E. G. Alexander for a breast tumor. Examination of the tissue removed by Doctor Alexander showed adeno-carcinoma. Six months later an ulcerated lesion about three centimetres in diameter appeared on the same breast. Doctor Deaver did a radical resection of the breast. The histological studies disclosed an associated carcinoma and tuberculosis in the same breast. In 1854 Rokitansky states that tuberculosis and carcinoma in the same individual, and more especially in the same organ, were incompatible. This has since been shown to be a fallacy.

DR. STEWART RODMAN recalled a case that occurred in his father's practice which has proven the association of tuberculosis and carcinoma in the same breast. The pathological diagnosis was confirmed by the late Doctor Copeland.

In so far as the treatment is concerned, most of those who have written on this subject advise resection of the diseased areas. As he had seen recurrences following this procedure, the speaker now practises and believes it to be the operation of choice—simple amputation.

TRANSACTIONS

OF THE

PHILADELPHIA ACADEMY OF SURGERY

STATED MEETING HELD DECEMBER 4, 1933

The Vice-President, DR. WALTER E. LEE, in the Chair

CALVIN M. SMYTH, JR., M.D., Recorder

TOXIC GOITRE WITH PARALYSIS OF EXTRA-OCULAR MUSCLES

DR. EDWARD J. KLOPP and, by invitation, DR. E. G. SHANNON, presented a man aged forty-five, a coal miner by occupation, who was admitted to the Jefferson Hospital, September 29, 1933, with the diagnosis of exophthalmic goitre. His chief complaints were nervousness, excessive perspiration, marked tremor of both hands, palpitation of the heart, staring of both eyes, diplopia and loss of weight. He has been a miner for the past twenty-five years, working from seven to eight hours per day, and has never been financially distressed. In August, 1932, there was an onset of marked nervousness with tremor of both hands and perspiration of the entire body. There was some gastric disturbance in the early part of August, during which he vomitted once. He was treated by a physician who diagnosed his case as "miners' asthma" on account of cough, expectoration and shortness of breath on exertion. He has not been able to work for the past five months.

The spinal fluid was practically normal. The Wassermann and Kahn reactions were negative. Metabolic rate on September 30 was plus 51; October 10, plus 29; October 18, plus 17; October 27, plus 15, and November 17, plus 25.

The patient was prepared for subtotal thyroidectomy by rest in bed and small doses of Lugol's solution. At operation under avertin, supplemented with nitrous-oxide anaesthesia October 31, 1933, nothing unusual was encountered. The most important symptom was the unusual paralysis of some of the extra-ocular muscles.

During his first illness in August, 1932, there was no clinical evidence of any ocular impairment. He was able to return to work in October. In January, three months later, he developed tearing of the right eye, followed by marked oedema of the right eyelid. With the subsidence of these symptoms he found he could not raise the right eyeball and that all objects appeared double except in the lower field. Five months later, the left eyeball was similarly affected and on October 2, 1933, both eyes exhibited an almost complete paralysis of the upward movements of both eyeballs, indicating an involvement of both superior recti muscles. In addition, the inferior oblique muscles were affected as no upward-outward rotation could be obtained. The inferior recti muscles being unopposed, it was noted that both eyes were turned definitely downwards, the right eye slightly lower than the left. (Fig. 1.) The left eye could be elevated slightly, the right eye apparently not at all. With binocular single vision obtained only in the lower field, it was necessary for the patient to tilt his head rather sharply backwards. Since the subtotal thyroidectomy on October 31, a little over a month ago, he feels

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that he can now bring his head nearer the erect position and still maintain single vision.

In attempting convergence, the right eye looked straight ahead while the left deviated in slightly. It is also interesting to note that with the eyes rotated to the extreme right the right eye turned downward, while with the

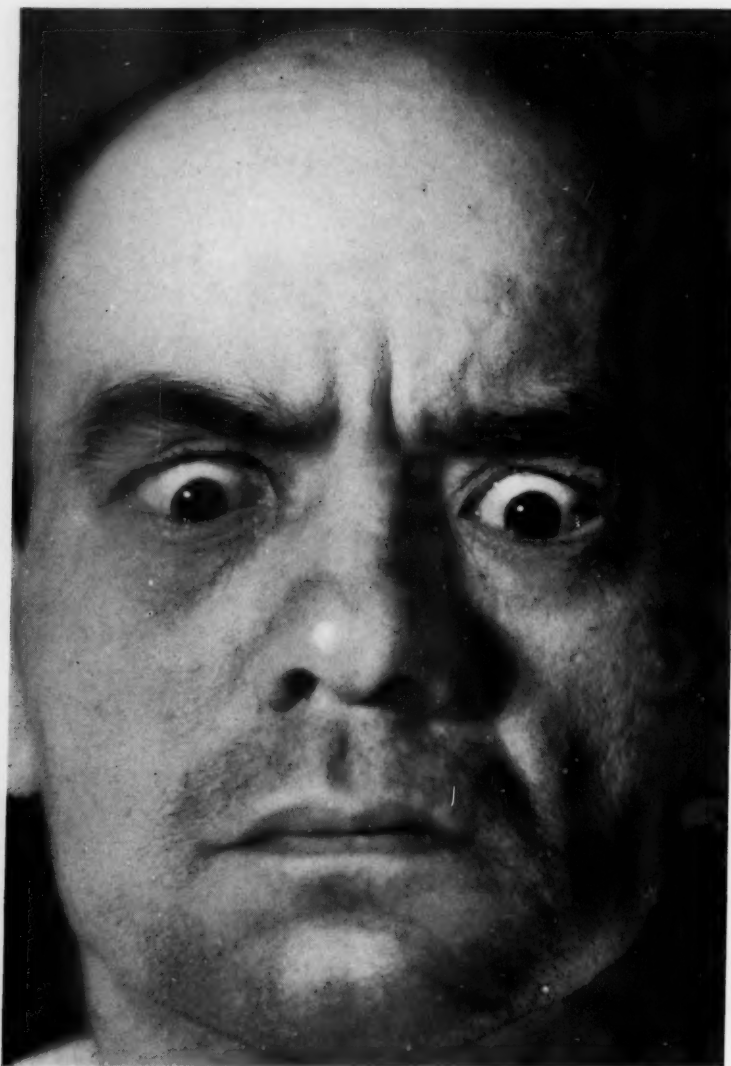


FIG. 1.—Toxic thyroid with paralysis of the superior recti and the inferior oblique muscles of both eyes with slight impairment of function of the externi and interni muscles.

eyes rotated to the left, the left eye was similarly deviated downwards, indicating an over-acting superior oblique muscle. In addition to the paralysis of the superior recti and inferior obliques, the function of the externi and interni muscles was slightly impaired. The fields for white and color were within normal limits and the eye-grounds, aside from some angiosclerosis, were negative.

TOXIC GOITRE WITH PARALYSIS OF EXTRA-OCULAR MUSCLES

The ocular symptoms that may accompany Graves' disease naturally draw the interest of the ophthalmologist. The symptoms were striking and important and it seems appropriate in connection with this report, before touching upon the unusual muscular complication in this case, to briefly enumerate them.

The explanations for the development of this condition are varied and as follows:

That it is due (1) to engorgement of orbital vessels; (2) to contraction of unstriated muscle fibres in the orbit, running from the equator of the eye to the orbital septum; (3) to deposition of fat in the orbit with œdema of ocular muscles. According to Foster-Moore⁶ the last explanation is the most tenable.

The speaker remarked that paralysis of the extra-ocular muscles in toxic goitre is rarely seen. Cases have been reported by Ballet,⁷ Liebrecht,⁸ Buschan,⁹ Manheim,² Lang and Pringle,¹⁰ West¹¹ and others, in some of which other cranial nerves have been involved. Palsy of the intrinsic muscles—sphincter of the iris and ciliary body—has not been recorded nor cases of associated movements of the eyes, with the exception of cases of paralysis of convergence by Schmidt-Rimpler¹² and Vossius.¹³ The occurrence of paralysis of, first, the right superior rectus and five months later, of the left superior rectus, would not support the view that this case was one of an associated paralysis of the superior recti. Bristowe¹⁴ reported a case of ophthalmoplegia externa in a young man three years after the symptoms of Basedow's disease had developed. Later loss of smell and taste occurred. Warner¹⁵ had reported a case of binocular external ophthalmoplegia with palsy of the facial and trigeminal nerves. Voss¹⁶ has reported two cases of palsy of the extra-ocular muscles in exophthalmic goitre. Bräin¹⁷ has recently reported five cases of enlarged thyroid and muscular disturbances. He offered no explanation of the relationship between the two conditions.

The interesting question arises as to the cause of the defective ocular movements. Various theories are advanced but the most acceptable is that of Foster-Moore,⁶ who believes that they are due to changes in the muscles rather than in the nerve of supply, a view, as he states, "which is supported by the distribution and by the fact that definite changes have been found in the muscles." This change, as he noted in one of his cases, was due to fatty infiltration of muscles.

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¹⁴ Bristowe: Brain, p. 313, 1886.
¹⁵ Warner: On Ophthalmoplegia Externa Complicating a Case of Grave's Disease. Med. Times and Gaz., p. 540, 1882; Lancet, vol. 2, p. 104, 1882.
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DR. THOMAS A. SHALLOW remarked that one could hardly believe that the same lesion which produced the bone changes in this man was responsible for the blindness and deafness. When we consider those factors about the eyes and ear, we are struck by this fact that in oxycephalus, which is a condition known as tower head, there is an early fusion of the sutures at the base of the skull. As a result, the subsequent development of the brain makes pressure and destroys some of the basal structures, particularly the first, third, fourth and sixth nerves. This belief is fortified by Doctor Keeler's Baranay test and by Doctor McAndrew's study of this child's eyes. There is quite a dispute about the consistency of the bone. Some agree that the bones are soft, others say that they are hard, and a review of the pictures shown by Doctor Davis indicates there has been softening at some time. There is no history of fracture, so here we have a condition which resembles Paget's disease and the reports of other cases show there is only bending as in this patient, but there is softening and, at a later stage, hardening of the bones.

When we consider the pathology in osteitis fibrosa cystica, which is caused by hyperparathyroidism and find that this produces osteoporosis and cystic change in the bone, we cannot help, by comparison, to classify osteopetrosis as hypoparathyroidism. since excessive lime-salt deposits are found in this disorder.

SOLITARY CYST OF THE FALCIFORM LIGAMENT

DR. HENRY K. SEELAUS remarked that among the 107 reported cases of solitary non-parasitic cysts of the liver, only three have been recorded as occurring in the hepatic ligaments, two in the falciform ligament, one by Dujarrie and the other by Wakely and MacMyn, and the third, in the round ligament by Bevan. The present report constitutes the fourth recorded case.

The patient, a girl, aged seven, was admitted to the children's ward of the Jefferson Hospital in the service of Doctor Bauer, July 11, 1933, with a diagnosis of hepatic cirrhosis with ascites. The present illness began three years ago with an enlargement of the abdomen which had gradually but progressively increased. In November, 1931, she was operated upon in another city, the operative note being that the liver was increased to ten times its normal size with an accompanying ascites. Nothing further was done and

SOLITARY CYST OF THE FALCIFORM LIGAMENT

the incision was closed without drainage, healed without complications and the patient sent home in ten days. Following her discharge from the hospital the abdominal enlargement increased and she was referred to the Jefferson Hospital. On physical examination the abdomen was markedly enlarged, distended and rounded, the superficial veins, including those of the umbilical region, being very much dilated. There was fluid present, the characteristic wave being elicited. An X-ray film of the abdomen confirmed the findings regarding fluid but there was so much fluid present that it prevented any further interpretation regarding any abdominal masses. A film of the pericardium and heart was negative for any evidence of Niemann-Pick's disease. The bromsophalein retention study of the liver indicated normal hepatic function; there was a positive indirect van den Bergh reaction and the quantitative van den Bergh study was .42 mg. per 100 cc. of blood. Under the impression that we were dealing with an instance of juvenile hepatic cirrhosis with ascites the abdomen was opened under ether anaesthesia through an upper Moynihan incision. Instead of finding an enlarged liver with ascites we encountered a large cyst adherent to the under surface of the liver and the great omentum. Being mindful of Wangensteen and Scott's work on shock following the sudden removal of large amounts of fluid from the peritoneal cavity we incised the fibrous wall of the cyst and with the aspirating set evacuated the cyst by gradual decompression, removing 3,850 cc. of a brownish, muddy fluid. The collapsed cyst was then easily shelled out from the folds of the falciform ligament and the under surface of the liver. The gall-bladder and the extrahepatic bile-ducts were entirely separate from the cyst and after its removal the two leaflets of the falciform ligament were brought together with a few interrupted catgut stitches. The oozing from the raw surface of the liver was controlled by pressing a hot moist gauze pad against it. The abdomen was closed with one cigarette drain carried down to the hepatic area. The patient reacted well from the operation and was discharged from the hospital three and a half weeks afterwards.

The fluid contained albumin and blood, no bile, a few pus-cells and many red blood-cells and debris.

Pathological Examination by Doctor Crawford.—Specimen consists of a cystic mass which has been emptied of its contents weighing 575 Gm. and measuring 19 by 17 by 15 cm. The external surface is white, smooth and glistening, and there are several ragged areas about 2 cm. in diameter, scattered over the surface. The cyst wall varies from $\frac{1}{4}$ cm. to $1\frac{1}{2}$ cm. in thickness. The wall is composed of a very dense grayish white tissue. The inner surface is smooth and covered with a slimy, brownish-yellow material.

Histology.—Examination of sections from the wall of the cyst reveals that it is composed of very dense fibrous tissue; in some places, the tissue is hyalinized. No other structure such as muscle tissue is observed, and in the number of sections examined no evidence of an epithelial lining to the cyst could be demonstrated.

TRANSACTIONS

OF THE

PHILADELPHIA ACADEMY OF SURGERY

STATED MEETING HELD JANUARY 8, 1934

The President, DR. WALTER E. LEE, in the Chair
CALVIN M. SMYTH, JR., M.D., Recorder

FRACTURES OF THE LOWER END OF THE HUMERUS

DR. WILLIAM BATES read a paper with the above title for which see page 1007.

DR. IRWIN E. DEIBERT said after reducing these fractures under the fluoroscope an attempt is made to throw them out of position by allowing the arm to hang loosely, thus ascertaining whether or not the fragments will become easily displaced. Once the fragments are placed in good position it is more or less difficult to throw them out of alignment. A simple dressing is applied in order to permit early motion, plaster being rarely used in this type of injury because of the tendency always to leave plaster on too long. Early motion, moving the part through the widest angle without the production of pain and supporting the fracture site is certainly productive of good results. If satisfactory reduction is not obtained immediately by simple methods an open operation should unhesitatingly be undertaken. It is the delay of an open procedure that is quite often the cause of a deformed or poorly functioning arm.

DR. HARRY E. KNOX remarked that in fractures involving the joints, particularly in the young and the aged, surgeons are prone to over-treat patients. In children with fractures of the lower end of the humerus and in those involving the elbow, fixation after reduction should apply until swelling has subsided and disappearance of pain, which usually requires from ten days to two weeks. The arm should then be removed from acute flexion to right angles, being maintained only by a sling, passive motion instituted, and active motion encouraged. By the end of three weeks the child will usually have little interference with function and by the end of four weeks can usually be discharged.

Complete reduction, while highly desirable, is by no means always necessary or possible for a good functional result. These fractures are to be treated individually and not to be viewed from a "standard." While the ideal should be the goal, it is not always possible to get a "cabinet maker's result."

DR. WILLIAM JOHN RYAN has never seen a supracondylar fracture which was caused by a fall on the outstretched hand. Those he has seen were caused by falling directly on the elbow, or, the elbow was struck by another

FRACTURES OF THE LEG BELOW THE LOWER THIRD

object while the individual was falling. In "T" or "Y" fractures, one frequently has wide separation of the lower fragments and Doctor Ryan has not been able to reduce them without open operation. It is necessary to bring the two lower fragments together with a screw or bolt or wire and then align them as one fragment with the upper.

DR. I. M. BOYKIN said that the mistake is often made in supracondylar fractures of attempting to reduce them without deep anæsthesia. The unreduced fracture is then dressed in acute flexion, resulting in circulatory impairment with swelling and vesication. After swelling has taken place it is futile to make further attempts at closed reduction. Recently the speaker has had under his care three such cases. They were dressed in full extension and an open reduction done when the swelling had subsided. In none of these cases was mechanical fixation necessary. In the post-operative care it is important to institute early weight carrying. This is much more preferable to active or passive motion and causes no pain.

FRACTURES OF THE LEG BELOW THE LOWER THIRD

DR. ADOLPH WALKLING read a paper with the above title for which see page 1009.

DR. LOUIS D. ENGLERTH said that Doctor Walkling has covered very well the treatment of the non-displaced simple fractures with which the speaker agrees.

In the simple fractures with displacement early reduction is paramount. These fractures should be classed as emergencies. Reduction is often easily obtained early, prior to swelling and fixation of the tissues by hæmorrhage, coagulation, and shortening. Anæsthesia is necessary as a rule. A fluoroscope is of distinct advantage. Following reduction, fixation in proper anatomical position is accomplished by means of a well-fitting, properly padded plaster case extending from the toes to above the knee. This is cut anteriorly or better still on both sides into an anterior and posterior shell. This allows inspection at any time, subsequent massage and manipulation, and can easily be shortened to below the knee at the proper time. One does not wait for swelling to go down. Early adequate reduction as an emergency treatment minimizes swelling. A plaster case splint is no more conducive to damage than any other method of fixation. Damage results from improper application and after care. Doctor Englerth uses the fracture box occasionally, usually as a preliminary splint and in patients kept in bed for some other reason.

Failure of reduction calls for more active treatment, preferably some form of traction most effectively obtained in this region by the skeletal method. Failing in this one considers open operation, the method of fixation depending on experience, judgment and type of fracture.

Treatment of compound fractures is always much discussed. Every compound fracture case should receive tetanus and gas bacillus antitoxin to be repeated if necessary. The speaker confessed that most of the time it is im-

possible for him to tell whether a compound fracture is from within or from without and he believes that this is academic rather than practical and from a clinical standpoint pays very little or no attention to it.

Every case of compound fracture is an emergency and should be treated as such. The term compound fracture centers one's interest on the bone whereas other considerations are of equal importance. The treatment which he practices is as follows: The patient is prepared under anaesthesia. The wound is protected with sterile gauze and the external surface is cleansed with soap and water and shaved. After further cleansing and sterilization the wound itself is thoroughly cleansed, irrigated, and mechanically and chemically sterilized, this being the most important phase of the treatment. Débridement and excision of the edges of the skin is practiced. Considerable time is devoted to the process.

At times the question of amputation arises. Under no circumstances is this to be performed in the presence of any evidence of circulation in the foot and even in the absence of circulation the patient should be given the benefit of the doubt and several days allowed before amputation is considered. No harm can be done in waiting. After thorough cleansing and débridement, further treatment is carried out. If, as often in this region, the ankle-joint is open, it should be closed after thorough irrigation and sterilization.

We now centre our attention upon the fracture. If reduction can be accomplished and maintained, that is all that is necessary. However, if reduction cannot be easily maintained some form of traction may accomplish the purpose. If this is not feasible, the fracture should be fixed by some recognized method such as plating, wiring, nailing, suturing, the use of clamps or bands. Judgment as to the method is necessary.

Attention is now centred upon the wound itself. If sufficient tissue is present the wound is closed without drainage. This requires considerable judgment. By a relaxing incision in some portion of the leg one is sometimes able to pull over sufficient tissue to cover the injury to the bone. A voluminous dressing is applied after closure has been accomplished. A large case is then applied extending above the knee down to the toes with the parts in perfect anatomical position. At times the Thomas splint is used.

If closure of the wound is impractical one selects a recognized method of treatment, either that advanced by Orr, with which he has had favorable experience, or by means of Dakin's treatment or some other antiseptic treatment until the wound closes. In some cases in which the wound has been allowed to remain open plastic surgery or skin grafting is later indicated.

DR. THOMAS RYAN said that he uses the fracture box at the Misericordia and Fitzgerald Mercy Hospitals as an emergency dressing only. Reduction is performed within twenty-four hours, under nitrous oxide or novocaine, and then a plaster case is applied, which is split down the centre and a bandage applied to keep it intact. He does not believe any injury can occur from early application of a case; in fact, it is very beneficial and relieves the

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patient's pain early. Regarding the period of time at which these patients should bear weight he does not believe that one can set any definite time but must be guided by union of the fracture based on X-ray. The sprain fracture, to his mind, should be placed in a case on account of the injury to the lateral ligaments of the ankle-joint, which requires four to six weeks for healing. The patient should not be allowed to walk early on this account. As far as the Steinman pin is concerned he has never found it necessary to use it in fractures below the lower third. Rather than raise the inner sole of the shoe it is better to apply a steel arch which supports the arch and the internal aspect of the foot.

As far as compound fractures are concerned Doctor Ryan does not feel that it matters whether they are from within out or from without in. Débridement to remove devitalized tissue and this procedure should be instituted when the amount of devitalized tissue associated with the fracture warrants it. The Orr treatment far excels any other form of treatment for compound fractures that has yet been advanced.

DR. IRVINE M. BOYKIN remarked that one of the prerequisites to the treatment of fractures generally is an understanding of the mechanism by which the fracture is produced. This is particularly true of the fractures in the lower third of the leg. With the exception of those fractures due to direct violence, the fractures of the lower third above the ankle are produced by the same forces which bring about fractures of the ankle, differing only in degree.

The most comprehensive classification of these fractures is one based on the mechanism which produces them. (Ashhurst and Bromer.)

(1) Outward rotation of foot (60 per cent.). Spiral fracture of fibula. Fracture of internal malleolus. (2) Fibular flexion (20 per cent.). Fracture of internal malleolus. Fracture of fibula in lower fifth. (3) Tibial flexion (13 per cent.). Fracture of external malleolus. Fracture of internal malleolus. (4) Landing on foot from height. T or Y fracture of tibia.

Fractures in the lower third, except those due to direct violence, occur while the patient is walking. The foot is caught, the body keeps moving or the individual slips and falls on his foot.

The diagnosis before swelling occurs can be made by digital examination, after swelling has occurred an X-ray is necessary.

As to the treatment of these fractures the speaker's feeling is that the fracture box has no place in the treatment of fractures in the lower third of the leg except perhaps as a splint for transportation. Any fracture which can be treated by this method can better be treated by the application of an adhesive-plaster boot. The objections to the use of the fracture box are:

(1) There is no extension. (2) It does not maintain reduction properly. (3) When opened for any purpose further displacement of fragments tends to occur.

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A preferable method for the treatment of these fractures is one which:

(1) Permits reduction without anaesthesia and without manipulation. (2) Is ambulatory and permits weight bearing. (3) One which secures union with free motion of the ankle-joint. (4) One which gives the shortest period of disability.

These points of advantage are all embodied in the Delbet method. No anaesthesia is required, reduction is secured through weight extension without manipulation. After the first week or ten days the patients become ambulatory, gradually bearing more and more weight. When union is firm and the splint can be removed it is found that the patient has full flexion and extension of foot. The period of disability is shorter. It has been statistically shown that the average period of disability in fractures of the lower third of the leg is five months. Ashhurst and Crossan in summarizing the end-results of 120 cases treated by Delbet's method at the Episcopal Hospital found that the period of disability was reduced to three and a half months.

The method has some disadvantages, *viz.*: The splints are difficult to make, the method is applicable only in hospital practice and requires a certain amount of skill. It is not applicable, except in rare instances in compound fractures, and in those cases with posterior dislocation of the astragalus. In spiral fractures of the middle of the lower third with posterior sagging of the lower fragment it is again not applicable. In the exceptions just mentioned we use skeletal traction in conjunction with molded plaster splints. When some union has taken place and the soft parts permit, the Delbet splints can then be applied.

Certain definite precautions must be taken to obtain the best results and prevent complications in using the Delbet method. The splint is applied as soon as the patient is admitted to the hospital. Reduction is obtained and maintained by weight extension, the splints being applied with the extension on. When the plaster has set the extension is removed, the leg elevated on pillows and ice applied to control swelling. Active motion of the ankle- and knee-joint is carried out daily. As the swelling subsides the splint becomes loose and must be reapplied before the patient is permitted to walk. When walking is permitted the patient is instructed in the use of crutches and in the necessity of elevating the limb when not in use. In many instances it is necessary to reapply the splint several times before it can be removed entirely. Pressure necrosis can be prevented by the proper application of the splint and the proper instruction to the patient as to how to care for his leg. The use of a special shoe following the removal of the splint is of prime importance in preventing flat feet.

It is obvious in the teaching of undergraduate students that other methods of treatment must be taught them because as previously stated Delbet splint is applicable only in hospital practice. Other methods which may be taught and which can be applied in home or office are:

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(1) Boehler's splint, which is a circular case in which is incorporated a steel loop which passes beneath the instep and on which weight is borne.

(2) Molded plaster splints can easily be applied under any conditions. The method has the disadvantages, however, of not permitting weight bearing.

(3) Split case is also a method which may be used.

Regardless of the method of treatment which one selects we will all agree that the earlier these fractures are reduced and splinted, the better our results.

DR. ELDRIDGE L. ELIASON said that immediate reduction means the earliest possible moment that reduction can be accomplished with safety to the patient. Two years ago the speaker analyzed some facts and figures on the time that elapsed between the ultimate reduction of the fracture and the time of the accident, and the average time in the long-bone shaft fracture was twelve hours. The man who applies the Delbet splint and does not keep his patient under close observation is going to get in trouble. This brings up the question of individuality in treatment of the fractures. One man should supervise the care of a fracture; not that he has to take care of each fracture each time it is seen, but his concepts of how it should be done should be transmitted to the man who looks after it, and he himself should see that fracture periodically. In the speaker's experience, most of the patients with bad results, such as they see at the Philadelphia General Hospital, give the history of being reduced, only after the swelling has been allowed to subside, which may be three days or two weeks and then sent out to be treated by a second person who does not take the same interest as the man who reduced the fracture. One of the speakers said the cabinetmaker's reduction is not necessary and should not be attempted. Literally, this is true. It is also true that the better the approximation the better function one can generally expect. Experience tells us many times that an imperfect reduction with a live patient and functioning limb is better than a cabinetmaker's reduction with loss of function due to injudicious open reduction.

Today emergency reduction is advocated. This means reduction at the very earliest possible moment that the safety of the patient permits. It means that the surgeon, the X-ray man, and if needed, the anæsthetist, get on the job, at any hour, day or night, Sunday or holiday.

One of the essayists advocates the use of the fracture box as a method of treatment. This is a very poor appliance and personally Doctor Eliason has not used one for more than twelve years. He regards it as a museum piece. The plaster case mentioned is a dangerous dressing in average hands. The Philadelphia General Hospital is the melting pot, or rather the clearing house, for the poor results in the other forty-three hospitals in this vicinity and we see some terrible wrecks, deformities and pressure sores from this type of fixation.

The ambulatory Delbet dressing has doubtless given good results in the hands of Doctor Crossan and Doctor Boykin, but it is a dangerous dressing applied by the average man with little experience.

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DR. HUBLEY R. OWEN said that he made some drastic statements in his paper concerning the faulty management of fractures in some of the hospitals. He could substantiate these statements. He sees some very bad results of fractures treated by other surgeons. Fortunately for him, no other surgeon sees his poor results because almost all of them occur among the policemen and firemen who must return to him for subsequent treatment. The Philadelphia General Hospital is the dumping ground for fracture failures.

Regarding Doctor Walkling's remarks concerning the use of the fracture box, it is Doctor Owen's personal belief that the fracture can be treated equally as well with a pillow splint. Far too often the fracture box is used for fracture of both bones of the lower leg, a procedure which is inexcusable. He wished to emphasize again the importance of a weight-bearing splint for fractures of both bones of the lower leg. In the past two years he has had thirteen cases of fractures of both bones of the lower leg occurring among the motorcycle patrolmen. Several of these were severe compound fractures. As soon as is feasible these men are allowed to employ weight bearing with an ambulatory splint and there has been no case of delayed union or non-union.

DR. DAMON B. PFEIFFER remarked that with due deference to the methods so carefully described by Doctor Walkling, and granting that with care excellent results can be obtained by this plan, he still feels that the time has come to discard the fracture box. The profession has now pretty well agreed that fractures call for immediate definitive treatment and in our hospital work it is uncommon not to be able to treat fractures of the leg on this principle, the exceptions being the presence of complicating injuries and poor general condition and those cases received too late for effective primary treatment. Some form of plaster case can be employed at once in the majority of fractures of the leg unless there has been large vessel injury with immediate and excessive swelling. In the ordinary case the danger of constriction by circular case has been greatly exaggerated. Still it exists and requires discretion and observation on the part of the surgeon. No method is fool proof so that the necessity for care and adequate precaution cannot be alleged as a contra-indication for the use of a method which gives the best result in competent hands. Only by proper fixation can good position be maintained. The expert use of plaster best fulfills this indication. While the speaker already indicated that he does not share the fear of circular casings, it is nevertheless true that at times the Delbet plan of splinting or the incorporation of pins for skeletal traction in the case are productive of results superior to the ordinary circular case. When these dressings are inapplicable, he confesses to a preference for the pillow splint over the fracture box. It is often possible to secure and maintain excellent reduction by the pillow splint in fractures of the lower portion of the leg, especially the Potts' fracture. It is more comfortable than the box and its soft elastic pressure tends to distribute and reduce the swelling.

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Now in regard to primary closure of compound fractures. It is true that in competent hands excellent, even brilliant results, are usually obtained, especially in fractures received in industrial plants where anaërobic organisms are not plentiful. It is equally true that in street and farm wounds, this method, however and by whomsoever carried out, is dangerous and that the serious nature of the failures more than counterbalances one's brilliant successes. There is a factor here which is uncontrollable by the surgeon, namely, the possibility of contamination of devitalized tissues and spaces by highly virulent organisms, and débridement, while it lessens the danger of infection, does not always prevent it. One does not need to mention the serious consequences to life and limb entailed by infection of a closed compound fracture.

For a number of years Doctor Smyth and the speaker have been greatly interested in the Orr plan and our results have been greatly improved. We have now had about 145 cases. There has been no disaster attributable to the method. There has been no tetanus or gas gangrene. There have been no deaths. One leg has been lost; that of an extensive compound fracture dislocation of the ankle in which the circulation was compromised and at the operation the joint could not be entirely closed. Gangrene and infection resulted and amputation was required but the patient survived with a good lower leg stump. Doctor Pfeiffer felt that he must restrain himself from speaking too enthusiastically of the Orr method for he is aware that there are many surgeons who do not welcome or actually oppose this plan of treatment. We can only say that in our hands it has seemed to solve the problems of compound fractures in most locations.

FRACTURES OF THE SHAFT OF THE HUMERUS

DR. CALVIN M. SMYTH, JR., read a paper with the above title for which see page 1013.

DR. V. W. MURRAY WRIGHT said it would be better for humanity if bones were rectangular on cross section rather than circular. X-ray pictures of a fracture are usually confined to the local site and rarely take in the entire length of the long bone which would permit a comparison of the planes at either end. This latter procedure would enable one to determine readily if internal or external rotation had occurred. Consequently, the use of the Jones' humerus traction splint may result in a limitation of external rotation in a healed fracture case because the lower fragment of the humerus was internally rotated as the forearm swung across the chest. As internal (or external) rotation of a round bone cannot be observed ordinarily in a röntgenogram it is necessary for us to watch out for and pick it up clinically.

DR. BENJAMIN LIPSHUTZ said that in the reduction of supracondylar fractures, traction is best carried out with the elbow flexed and the forearm in position of pronation. In this way one can relax the pronator and other muscles arising from the medial epicondyle of the humerus; frequently a good reduction is not obtained unless the pronator is relaxed.

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Some fractures of the humerus following reduction are well maintained by a plaster bandage from the wrist to axilla with forearm flexed to a right angle. A ring of wire is incorporated in the upper side of the cast at the wrist, and through this the suspending sling is threaded which prevents the sling from sliding. This treatment does not immobilize but restricts motion, and weight of the case applies traction while the patient is standing or sitting.

DR. L. K. FERGUSON said that in his experience reductions of the fracture of the shaft of the humerus were much easier if the patient be allowed to sit up during the reduction. In this position the weight of the arm is often sufficient to overcome shortening and produce reduction. When this position is employed in conjunction with the aid of the fluoroscope and local anæsthesia reduction is often a simple matter. The coöperation of the patient under these conditions makes it much easier to apply the dressing and gives more satisfaction after application.

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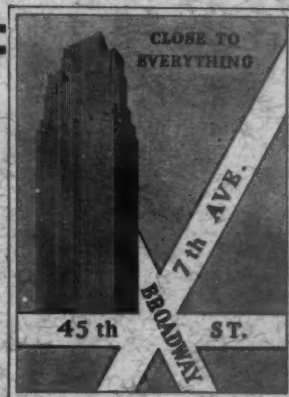
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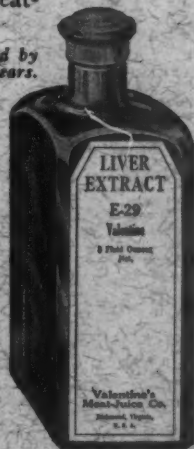
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